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Seventh Saranac Symposium

Page 111 missing (partial page 111 noted taken from page 111)

Pages 554 thru 564 missing

Pages 596 thru 621 missing

Pages 634 thru 678 missing

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✓ Abbey, R. G. , MR. Safety Engineer,
General Refractories Co.

✓ Adams, Charles T. Edwards, New York

✓ Aggott, Albert G. MR. Claim Department 142 Berkeley St.
Amer. Mut. Lia. Ins. Co. Boston, Mass.

✓ Albaugh, Roscoe T. MR. Republic Steel Corp. 1912 Scranton Road
Cleveland, Ohio

✓ Alexander, P. P. Mr. President Beverly, Mass.
Metal Hydrides, Inc.

✓ Alexander, W. A. Dr. Dept. Metallurgical Engin. Toronto 5, Canada
University of Toronto

✓ Amberson, J. Burns, Jr. m.d. Bellevue Hospital 27th St. & First Ave.
New York 16, N. Y.

✓ Ames, Edward C. MR. Owens-Corning Fiberglas Corp. Toledo 1, Ohio

✓ Ancira, Erasmo Gonzalez Av. San Francisco 345 Col. del Valle,
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✓ Anderson, Robert MR. Norway, Michigan

✓ Andrus, Stephen B. Jr. 40 Warren St.
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A. Arn, V. P. , MR. National Industrial Sand Assn. Munsey Bldg.
Washington, D. C.

✓ Ames, W. F. MR. Manager, Compensation & Safety Bethlehem, Pa.
Bethlehem Steel Co.

HOTEL SARANAC RESERVATIONS

| Name | 21 Sun | 22 Mon | 23 Tues | 24 Wed | 25 Thurs | 26 Fri | 27 Sat | Reser. Made By | Arriv- ing By |
|--|-----------|-----------|------------|-----------|-------------|-----------|-----------|----------------------|---------------------|
| Albert, Roy E., M.D. <u>Wife</u> | ✓ | 2 PM | X | X | X | X | | LRB | car |
| Ashe, Harry B. | ✓ PM | X | X | X | X | X | | LRB | car |
| Aggott, Albert G. J. B. SKINNER | | X | X | X | X | X | | car | |
| Baetjer, Anna, M.D. | ✓ X | X | X | X | X | X | | LRB | |
| Bamberger, Paul J., M.D. W.T. ROBERTS | ✓ PM | X | X | X | X | X | | LRB | car |
| Barnako, F. R. | ✓ | | | | X | X | X | LRB | |
| Beattie, J., Dr. J.F. KNOX | ✓ AM | X | X | X | X | X | | LRB | car |
| Bell, Robert F., Dr. J.J. WARING | ✓ 4:40 PM | | | | | | | LRB | |
| Bellamare, Ida Tourangeau | X | X | X | X | X | X | | LRB | car |
| Benedict, Karl L., M.D. | ✓ DPM | X | X | X | X | X | X | LRB | car |
| Broddkin, Henry Dr | | X | X | X | X | X | | car | |
| Bowditch, Manfred | ✓ X | X | X | X | X | X | X | LRB | |
| Brennan, J.H.L., M.D. E.W. FLAHIFF | ✓ 9 PM | X | X | X | X | X | | LRB | |
| Brieger, H., M.D. | ✓ | X | X | X | X | X | | LRB | |
| Connolly, Wm. L. Wife | ✓ | | | | X | X | | LRB | |
| Havis, F. T. M. S. W. T. | 21 | X | X | X | | | | car | |
| Donovan, P.H. WIFE | ✓ | 7:10 AM | X | X | X | X | | LRB | |

| Name | 21 Sun | 22 Mon | 23 Tues | 24 Wed | Thurs | Fri | Sat | Reser. Made by | Arriv ing by |
|---|-----------------|---------------|------------|-----------|-------|-----|-----------|----------------------|--------------------|
| Drinker, Philip, Prof. | ✓ | 2. | 419 X | | | | | | |
| Eddy, George P., M.D. | ✓ | X | X | X | X | X | | Coif | |
| D. N. E. P. Eggle | | | | | X | | | Coif | |
| Eisenbud, Merrill + Mrs | ✓ | 9 AM | X | X | X | | | Coif | |
| Fine, Melvin L. | ✓ | | | X | X | X | X | Coif | |
| Flahiff, E.W. J. H. L. BRENNAN | ✓ 21 | AR | X | X | X | X | | LRB | |
| Fleming, B. J. R. D. TELLEFSON | ✓ X | X | X | | | | | Coif | |
| French, James M.D. | ✓ AR | AR X | X | X | | | | Coif | |
| Fritz, Frank, Dr. Twin | ✓ PM | X | X | X | X | X | to 28 (2) | Coif | |
| Gentholtz, A. J. | ✓ | | | | X | X | | Coif | |
| Greenburg, L., M.D. WIFE | ✓ X | X | X | X | X | X | X 9/28 | LRB | |
| Gregoire, Fernand, M.D. J. A. VIDAL | ✓ X | X | X | X | X | X | X | LRB | |
| Hamlin, L. E., M.D. WIFE | ✓ 9/20 PM | X | X | X | X | X | X | LRB | Car |
| Hanson, Harry A., M.D. J. H. STERNER | ✓ 9/20 NOON | X | X | X | X | X | | LRB | Car |
| Hayes, D. L. | ✓ | | | X | X | X | X | Coif | |

| Name | 21 Sun | Mon | Tues | Wed | Thurs | Fri | Sat | Reser. Made by | Am in t |
|--|------------|----------|--------------|-----|-------|-----|-----|----------------------|---------------|
| Hazard, W. G. | ✓ | 7:10 AM | X | X | | | | Himself | |
| Herman, Nathan, M.D. WIFE | ✓ X | X | X | X | X | X | X | Cox | |
| Hill, James L. DOUBLE | ✓ | | | X | X | | | LRB | |
| Hueper, W. C., M.D. | ✓ | 10:17 AM | X | X | X | X | | Himself | |
| Hussey, Raymond, M.D. See Long | ✓ 9/19 | X | X | X | X | X | X | Cox | |
| Irvin, E. A., M.D. | 9/20 | X | X | | | | | | |
| Johnstone, R.T. | 9/19 | X | X | X | X | X | 27 | | w/Bar |
| Jackson, H. M. K.W. SMITH | ✓ X | X | X | X | X | X | | LRB | |
| Kelly, Emmet, M.D. | ✓ | X | [TENTATIVE!] | | | | | Cox | |
| Kerr, Lorin E., M.D. DOUBLE | ✓ 10 PM | X | X | X | X | X | X | Cox | |
| Kienzle, T. C., M.D. DOUBLE | ✓ | A.M. | X | X | X | X | | Cox | |
| Kline, E. M., M.D. | ✓ | 7:10 AM | X | X | X | | | Cox | |
| Knox, J. F., Dr. Beattie | ✓ X | X | X | X | X | X | | Cox | |
| / Canceled | | | | | | | | | |
| Krieger, H. L. | X | X | X | X | X | X | X | Cox | |
| Lange, Paul C. F. SCHLEUTER | ✓ X | X | X | X | X | X | X | LRB | |

| Name | 21 Sun | 22 Mon | 23 Tues | 24 Wed | 25 Thurs | 26 Fri | 27 Sat | Reser. Made by | Arri- ing by |
|--------------------------------------|-----------|-----------|------------|-----------|-------------|-----------|-----------|----------------------|---------------------------------------|
| Lanza, Anthony, Dr. C.M. PETERSON | 9/19 | X | X | ? | ? | ? | | | |
| Lauer, D. J., M.D. DOUBLE | X | X | X | X | X | X | | Co | |
| Levin, M. L., M.D. | | | X | X | | | | LRB | |
| Long, Gillmore, Dr. | X | X | X | X | X | X | | LRB | |
| Lutz, E. F., M.D. WIFE | PM | X | X | X | X | X | | LRB | Car |
| Lynch, Kenneth, M.D. | | | | 7:10 AM | X | | | LRB | |
| Mallett, D. W., M.D. DOUBLE | 6 PM | X | X | X | X | X | | LRB | Dn/m |
| Martin, Dr. J. E. | 9/21 | X | X | X | X | X | X | | |
| Massie, E. W. DOUBLE | | X | X | X | X | X | X | Co | |
| McCahan, J. F., Dr. R. HUSSEY | 9/19 | X | X | | | | | | |
| W. J. Mc Connell | | | X | | | | | Co | |
| McCormick, W. E. | 7 PM | X | X | X | X | X | | LRB | |
| McGee, Lemuel, M.D. WIFE | | | | | 4 PM | X | | LRB | Car |
| Miller, Seward C.A. Irwin | Ro | X | X | | | | | | |
| Kenneth Moore | | X | X | X | X | X | | LRB | Cancelled LRB |
| Muenzer, J. A., M.D. | 9/17 | X | X | X | X | X | X | Co | 2 double Connell (Irwin) don |
| Pedroza, Dr. S. M. VERA | 9/20 | X | X | X | X | X | X | LRB | |

| Name | Sun | Mon | Tues | Wed | Thurs | Fri | Sat | Reser. made by | Arriv- ing by |
|--|------|---------|------|-----|-------|-----|-----|----------------------|---------------------|
| Peterson, C. M., M.D. <u>A. J. LANZAN</u> | 9/19 | X | X | | | | | | |
| Pinkerton, H. A., M.D. <u>WIFE</u> | X | X | X | X | X | X | X | LRB | |
| Pinto, Sherman, Dr. <u>DR. RONDE</u> | 5 PM | X | X | X | | | | LRB | |
| Richards, Paul S., M.D. <u>O. WISLEY</u> | X | X | X | X | X | X | | Conf | |
| Roberts, W. T., E.D. <u>P. J. BAMBERGER</u> | X | X | X | X | X | X | | LRB | |
| Rohde, F., Dr. <u>S. PINTO</u> | | 7:30 AM | X | X | X | X | | LRB | |
| Sander, O. A., M.D. <u>R. T. JOHNSTONE</u> | 9/19 | X | X | X | X | X | X | Conf | |
| Schleuter, C. F. <u>P. LANGE</u> | X | X | X | X | X | X | X | Conf | |
| Selby, C. D., M.D. <u>Werner</u> | 9/19 | X | X | X | X | X | X | Conf | |
| Shaver, C. G. <u>D. Y. SOLANDT</u> | 9/21 | X | X | X | X | X | | Conf | |
| Skinner, John B. <u>A. G. ACCOTT?</u> | | X | X | X | X | X | | Conf | |
| Smith, K. E., M.D. <u>H. M. JACKSON</u> | 6 PM | X | X | X | X | X | | LRB | Car |
| Solandt, D. Y., Dr. <u>C. G. SHAVER</u> | 9/21 | 2X | X | X | X | X | | Conf | |
| Sterner, James, M.D. <u>H. A. HANSON</u> | 9/20 | X | X | X | X | X | | LRB | Car |

| Name | Sun | Mon | Tues | Wed | Thurs | Fri | Sat | Reser. made by | Arriv- ing by |
|-------------------------------------|------------|--------|------|-----|-------|-----|-----|----------------------|-----------------------|
| Stratton, R. C. FAMILY | ✓ PM 10-11 | X | X | X | X | X | | Himself | Car Train Plane |
| STRAIN, MR. FRANKLIN | ✓ PM | 8-9 AM | | | | | | | |
| T-brock, H. E., M.D. DOUBLE | ✓ AR | X | X | X | X | X | | LRB | |
| Teilefsen, Donald B.J. FLEMING | ✓ X | X | X | | X | X | | Cor LRB 3/4 Car | |
| Tourangeau, Dr. DR. R. BALLMANN | ✓ X | X | X | X | AR | LV | | LRB | |
| Tiernon, J. Harry | | | | X | X | X | LV | Cor | |
| Tucker, Warren | | | | X | X | X | X | LRB | |
| Van Ordstrand, H. S. WIFE M.D. | ✓ X | X | X | X | X | X | X | | |
| Vera, Miguel, Dr. DR. S. PEDROZA | ✓ 9/20 | X | X | X | X | X | | Cor | |
| Vidal, J. A., Dr. DR. GREGOIRE | ✓ X | X | X | X | X | X | X | LRB | |
| Waring, J. J., M.D. R. BELL | ✓ 4 PM | X | X | X | X | X | X | Cor Plan | |
| Waters, Ted. | ✓ | | | X | X | X | | LRB | |
| Whipple, E. G., M.D. Twin | ✓ X | X | X | X | X | X | X | Himself | Car |
| Wiesley, Otto A. RICHARDS | ✓ X | X | X | X | X | X | | Cor | |
| Wyatt, John P., M.D. WIFE | ✓ 6 PM | X | X | X | | | | Cor | Car |

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+
Double
Check

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Participants
in
Seventh Saranac Symposium

1952

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Seventh Saranac Symposium
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Pulled -

After Bennett M. Lynch Paper

"Carcinoma of the Lungs
in Asbestos Workers"

Art -

Today's Times tells me that
you have accepted another post -
and will be leaving Sarnia Lake.
Where are you going?

I would very much enjoy
it if we could meet for lunch
next time you are in the city.

Ad best Bill

Pulled

TBI

Some Concepts of the
Biological Action of
Silence —

Tom
Dunkins
Paper

Symposium

Count.
understanding ~~Source~~

"Every excess causes a defect; every defect an excess Every faculty which is a receiver of pleasure has an equal penalty put on its abuse with every influx of light comes new danger"

Emerson

Foreword

PREFACE

Use some of Foreword of 7th symposium announcement
Include acknowledgement to companies who will pay for publishing the
introduce correction of papers and all
→ major discussion

The speakers gave their time
without compensation and
it is a pleasure to express
publicly our gratitude to
them —

Thanks to each and every
member of the staff — without
^{help} whom this symposium would not
be possible

The topics set down for discussion
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PULMONARY CANCER

Pulmonary Function

COMPENSATION

Pul. Function?

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SEPTEMBER 22

TO

SEPTEMBER 26

1952

THE SARANAC LABORATORY
of the
Edward L. Trudeau Foundation
Saranac Lake, New York

Preface (2)

Foreword

The Saranac Symposiums, of which this session is the Seventh, have been organized periodically for the discussion of health problems pertaining to workers exposed by inhalation to industrial substances. The subjects to be discussed at the Seventh Saranac Symposium will be limited but many have broad application. The topics will not be restricted to individual companies or industries, nor will they concern only a single group such as the employer or the employee, the insurer or the insured. The problems to be considered involve a host of diverse interests — engineering, medical, legal, administrative, social. At the symposium representatives of each of those groups and interests will have the opportunity to explore and discuss common problems with the objective that industrial health be improved. If that objective is attained the symposium will be a successful one.

Monday, September 22

2:00 P.M. — 5:30 P.M.

**The Inhalation of Certain Industrial
Substances**

Chairman: THEODORE F. HATCH

Brief Reviews

Silica

Thomas M. Durkan

Asbestos

Arthur J. Vorwald, M.D.

Beryllium

Harriet L. Hardy, M.D.

Bauxite

C. G. Shaver, M.D.
and
Donald Solandt, M.D.

Discussion

New Synthetic Silicas

Philip C. Pratt, M.D.

The Fate of Inhaled Particulates

Merril Eisenbud

Discussion, led by

Theodore F. Hatch

Tuesday, September 23

9:00 A.M. — 1:00 P.M.

Pneumoconiosis in Coal Miners

Chairman: PHILIP DRINKER, Sc.D.

*Industrial Hygiene Studies of Coal Miners
in Two Geographical Areas in the United
States*

E. C. J. Urban

Discussion

Radiological Classification

Charles M. Fletcher, M.D.

Discussion

Pathology of Coal Workers' Pneumoconiosis

Arthur J. Vorwald, M.D.

Discussion

*Epidemiology of Coal Workers' Pneumo-
coniosis in Wales*

Charles M. Fletcher, M.D.

Discussion

Tuesday, September 23

2:30 P.M. — 5:30 P.M.

Pneumoconiosis in Coal Miners
(Continued)

Chairman: O. A. SANDER, M.D.

Pneumoconiosis in Coal Miners in Alabama
Louis Friedman, M.D.

Discussion

*Pulmonary Function Studies of Coal Miners
in Pennsylvania and West Virginia*
Hurley L. Motley, M.D.

*Pulmonary Function Studies of Coal Miners
in Wales*
Philip Hugh-Jones, M.D.

Discussion

Wednesday, September 24

9:00 A.M. — 1:00 P.M.

Pneumoconiosis and Pulmonary Cancer

Chairman: C. P. Rhoads, M.D.

**Philosophy of Biostatistics as Applied to
Environmental Pulmonary Cancer**

Morton L. Levin, M.D.

Discussion

General Review

W. C. Hueper, M.D.

Discussion

Asbestos Miners

Paul Cartier, M.D.

Discussion

Asbestos Weavers

Kenneth M. Lynch, M.D.

Discussion, led by

E. R. A. Merewether, M.D.

Group Photograph

MOENING

Wednesday, September 24

2:30 P.M. — 5:30 P.M.

Pneumoconiosis and Pulmonary Cancer
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Discussion

Experimental Pulmonary Cancer — General
Review G. Burroughs Mider, M.D.

Discussion

Pulmonary Cancer in Experimental
Exposures to Chromate Anna Baetjer, Sc.D.

Discussion

Pulmonary Cancer in Experimental
Exposures to Beryllium Arthur J. Vorwald, M.D.

Discussion

Thursday, September 25

9:00 A.M. — 1:00 P.M.

Clinical Aspects of Pneumoconiosis

Chairman: WILLIAM S. McCANN, M.D.

*Incidence of Clinically Manifest Diffuse
Obstructive Emphysema*

Leonard J. Bristol, M.D.

Discussion

*Pulmonary Function Studies in Men Exposed
for Ten or More Years to Inhalation of
Asbestos Fibers*

Fernand Gregoire, M.D.

Discussion

*Maximum Ability for Physical Work of
Moderate Duration Correlated with Various
Tests of Pulmonary Function*

George W. Wright, M.D.

Discussion

*Cardio-Circulatory Aspects of the Pneumo-
conioses*

William S. McCann, M.D.

Discussion, led by James J. Waring, M.D.

Thursday, September 25

2:30 P.M. — 5:30 P.M.

**Workmen's Compensation — Medical and
Legal Aspects**

Chairman: M. W. THOMPSON

*Concepts of Disability Under Workmen's
Compensation Statutes*

Theodore C. Waters

*Compensation for Disability and Death Re-
sulting from the Pneumoconioses*

George W. Wright, M.D.

George E. Meredith

Martin F. Hilfinger

Discussion, led by

Andrew Kalmykow

7:30 P.M.

Banquet — Hotel Saranac

Introduction of Guests — Arthur J. Vorwald, M.D.

Remarks — Francis B. Trudeau, M.D.

Leroy U. Gardner Memorial Lecture

Anthony J. Lanza, M.D.

Friday, September 26

2:00 P.M. — 5:30 P.M.

**Administration of Workmen's Compensation
Laws as They Relate to the Pneumoconioses**

Moderator: THEODORE C. WATERS

Panel Members:

James L. Hill
William L. Connolly
B. E. Keuchle
Earle T. Andrews
Lloyd E. Hamlin, M.D.

*Otto A. Worely
substituted*

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Workmen's Compensation Sessions

Theodore C. Waters

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*Summary of the Medical Sessions and
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SEVENTH SARANAC SYMPOIUM

THE SARANAC LABORATORY of the
Edward L. Trudeau Foundation,
Saranac Lake, New York

Monday
September 22, 1952

SEVENTH SARANAC SYMPOSIUM

THE SARANAC LABORATORY of the
Edward L. Trudeau Foundation,
Saranac Lake, New York

September 22, 1952
Session Commencing 9:45 A. M.

Transfer
to Forward

1.

ADDRESS OF WELCOME

ARTHUR J. VORWALD, M.D.

BY DOCTOR VORWALD:

Ladies and Gentlemen: The members of the Staff of the Saranac Laboratory and of the other departments of the Trudeau Foundation, are delighted to act as hosts to you who come here for the Seventh Saranac Symposium.

On behalf of those members, I am privileged to extend a most cordial welcome to you all, especially to those of you who have come from distant lands, to participate in this Symposium. You represent many different interests and include physicians and engineers, lawyers and insurance executives, specialists in compensation and labor matters, and representatives of management and of labor.

~~Now,~~ Although those interests are diverse, nevertheless, ~~those interests~~ ^{they} have a common objective, namely, to give the utmost in health and happiness in our way of life, as indicated in the Foreword of your printed program.

I wish, again, to impress upon you that the topics to be discussed at this Symposium must be limited. Those topics have broad application, and they are not restricted to individual companies or industries, nor do they concern only a single group such as the employer or the employee, the insurer or the insured. The ^{discussions} ~~talks~~ here, during the course of the week, will cover a wide range of subjects, which you will ⁱⁿ explore in friendly ^{manner} ~~discussion~~. As heretofore, this Symposium will give all of us the opportunity

to exchange ideas in an informal way, which will further our understanding and solution of problems pertaining to some aspects of pulmonary disease in the industrial worker.

We hope, sincerely, that your deliberations will benefit us all in our common objective, and that you will look back with pleasure and satisfaction to your so-journ here in Saranac Lake.

In opening this Symposium, it is my privilege to make certain announcements which I shall try to do every morning or periodically during the day, to keep you informed and to assist you in your stay with us.

As you realize, there is Registration at the John Black Room. We had hoped to register all before this session. However, shortly after nine thirty, we saw that was going to be impossible, because it would delay our meeting this morning. We thought it was more important to get on with our discussions than to have you register. However, we do hope that you will register sometime during the day.

Those of you who come to join us from outside of Saranac Lake, on registration, will receive white badges. Those of us who reside in Saranac Lake will receive colored badges, the green for members of the Trudeau structure, the pink for members of Saranac Lake medical profession, legal profession, and others who have an interest and will

be here, so that, should any of you wish to learn of things or wish to have help, ask one of us who have colored cards.

Automobile tags on registration: A number of you have come with cars, and as you will note, there are parking meters throughout the village. However, the Chief of Police, Chief Wallace, in the interest of this Symposium, has graciously waived the parking meter ordinance and, on registration, you will receive a red card, which we hope you will show on the inside of your windshield somewhere, so that the Police Department may see that card and not fine you for having parked overtime, so that that card will allow you to park anywhere in the village, as long as you wish, during the Symposium.

On Thursday night, we have a banquet, and we hope that, on registration, you will also buy your tickets for the banquet, because that is important, since we must notify the Hotel management as to the number who might be attending.

As to mail, incoming mail may be obtained either at or in the John Black Room of the Saranac Laboratory, by asking the Secretary, or by inquiry at the desk in the foyer of this building. Outside of the foyer, you will find a bulletin board on the wall where we will attempt or try to put notice for those of you who might be called to the phone or might have special delivery letters or other

things which should come to you.

The agenda, as you will note, is a crowded one and it begins every morning at nine o'clock. Then, it will go until twelve or twelve thirty, as you follow your agenda. Today, we have set aside two hours for lunch, because it's the first day and we thought that you would be looking for places to have lunch. On all other days, the lunch period is one hour and a half. Now, there are various eating places in town, which I know will make every effort to serve you as promptly as possible so that you can return here to this hall for the afternoon session. Then, too, there are many of you who are Elks and the Elks Club is up the street to your right, down the hill and then up a hill and right at the top, and it's a very delightful place. You are welcome to go there and also to invite your friends to join you, both for lunch and dinner in the evening.

The agenda is crowded, and we apologize for the lack of provisions for play, but the evenings should afford you a sufficient time to do some of the few things for relaxation which you wish. There are many of you who live or who are staying in accommodations on the outskirts of town and some of you living in those accommodations are without cars. You may call a cab, or you may go on the highway in the morning and start thumbing and should members of this

Symposium with cars pass by, I hope that they will honor those with cards on their lapels, who might be doing the thumbing.

You may visit the John Black Room of the Saranac Laboratory to see the exhibits, visit the other components of the Trudeau Foundation and also the Trudeau Sanitorium -- many of you know where those facilities are located. We do hope and urge that you will find time to visit these facilities and to explore some of the activities going on in the various departments.

That, then, brings us to the first topic on the Symposium, and in the discussions throughout the week, I do hope that those of you who participate will give your name and your location so that the stenotypist may record it for the record.

**DEFINITIONS AND CLARIFICATION OF TERMS
PERTAINING TO PNEUMONONIOSIS**

Moderator: Arthur J. Vorwald, M. D.

Panel Members: Anthony J. Lanza, M. D.
Leonard Greenburg, M. D.
O. A. Sander, M. D.
Richard Wagner
Charles M. Fletcher, M. D.

Discussion, led by A. J. Gorenstein, M. D.

BY DOCTOR VORWALD:

With that then, the first topic on the agenda for

DEFINITIONS AND CLARIFICATION OF TERMS
PERTAINING TO PNEUMOCONIOSIS

Moderator: Arthur J. Vorwald, M. D.

Panel Members: Anthony J. Lanza, M. D.
Leonard Greenburg, M. D.
O. A. Sander, M. D.
Richard Wagner
Charles M. Fletcher, M. D.

Discussion, led by A. J. Orenstein, M. D.

BY DOCTOR VORWALD:

With that then, the first topic on the agenda for

this morning concerns the Definitions and Clarification of Terms Pertaining to Pneumoconiosis, and it will be carried along by a panel, and you can see the members of the panel. Now, in planning the agenda for the Symposium, we were urged by many colleagues to include for discussion, the definition and clarification of terms which pertain to the inhalation of atmospheric substances and which have given and are giving rise to considerable debate.

Now, those colleagues obviously are somewhat disturbed by the lack of uniformity in usage and definitions of certain terms, particularly of those terms which lack clarification, and which permit a multitude of interpretations, often extremely unorthodox and frequently confusing.

Some colleagues, although desirous of clarification, evidenced considerable pessimism, for fear that discussion at this symposium, involving a large group of individuals, rather than a small panel, would fail to resolve many of the issues concerning definitions, usage, interpretation of terms, and the like.

Now, that pessimism is appreciated, but it was felt that that feeling should not deter us from placing the topic on the agenda, believing that much good would accrue if no more than to pave the way for action by some other group or some other panel. It is true perhaps, that our discussions here today concerning definitions and terminology,

will involve words, and as such, may be looked upon as being somewhat picayune, perhaps too academic, perhaps of little practical value, and too difficult to tackle, but words are tools with which we think, and we ought to try to select the most appropriate tools for the job which we are all trying to do.

Now, that job pertains to the inhalation of industrial substances and includes engineering, medicine, the legal phases, compensation and the like, so I'm sure that you appreciate the difficulties in planning a discussion of words and definitions and the like, which constitutes the topic for this session.

Should the session prove disappointing and non-productive, then your Chairman takes full responsibility for that failure. On the other hand, should it be a stimulating and successful one, then the Symposium will owe you a debt of gratitude.

The framework of our discussions on definitions and terminology, it seems to me, should concern such words as "pneumoconiosis", "inactive and active dusts", terminology for specific disease entities, and, in considering it, it seems to me that we should give or should approach it from two points of view perhaps: The medical point of view and the legal point of view, so keep those things in mind, and each of those points of view might be explored

NEW YORK UNIVERSITY-BELLEVUE MEDICAL CENTER

OF NEW YORK UNIVERSITY
INSTITUTE OF INDUSTRIAL MEDICINE

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Mailing Address: 477 FIRST AVENUE, NEW YORK 16, N.Y.
Telephone: MURRAY HILL 9-1580

September 8, 1952

Dr. Arthur J. Vorwald
Saranac Laboratory
Saranac Lake, New York

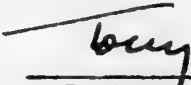
Dear Art:

Enclosed are my comments on the Panel. As you will note, they are very brief and written more or less in telegraphic form so that they may be expanded by suitable comments as the case may demand and would permit plenty of room for argument.

My comments on chrome cancer are also very brief. Do you want an advance copy of that too?

With best regards,

Sincerely yours,


A.J. Lanza, M.D.

AJL:lc
Enc.

PNEUMOCONIOSIS

A.J. Lanza, M.D.

September 22, 1952

10 a.m.

*Remarks
by
Panel
Member*

As a preliminary statement concerning this Panel it is important to determine what it is we are discussing. If we can agree on that we may hope to agree on terms.

We are concerned with occupational diseases of the lungs due to the inhalation of dust--occupational diseases which occur all over the world, which may cause disability and death, and which do not occur except as occupational diseases. The importance of these diseases has become recognized, as far as modern industrial conditions are concerned, within the past forty-five years. The particular disease which has especially attracted attention is what we now call silicosis.

About 1918 Landis stated that organic dusts--textile dust, vegetable dust, wood dust--did not cause the definite occupational lung disease which was becoming more and more recognized as the evidence of surveys of many industries began to accumulate, and ^{he} revealed that various diseases to which trade names had become attached were one and the same. The villain of this piece is silica--principally free silica and to a lesser extent various dusts of combined silica.

Almost everybody breathes silica in one form or another during his lifetime; everybody inhales water vapor. The one does not develop dust disease of the lung; the other does not drown. But when the dosage of either dust or water becomes too large for the respiratory system to cope with, unfortunate results follow. So to repeat: We are concerned with occupational diseases of the lungs caused by the inhalation of dust.

These diseases are of slow onset and are characterized by the formation of pulmonary fibrosis accompanied by a varying degree of dyspnea. Some of these diseases are further characterized by an increased susceptibility to infection, particularly by the tubercle bacillus. They present a radiologic picture which may be quite characteristic but which at times may also be confusing. However, these X-ray films of dust diseases of the lungs show a pathological involvement more or less evenly distributed throughout both lungs. The added factor of infection, when present, may confuse the diagnosis especially when the nature and extent of dust exposure ^{are} ~~is~~ not known.

We know a great deal about the occupational disease of the lungs due to the inhalation of ^{crystalline} ~~crystalline~~ silica. There is still much we do not know about silicosis, the term commonly applied to this disease, in spite of the fact that a tremendous amount of research has been carried on. We know less about the action of asbestos and still less about the results of inhaling diatomaceous earth. We may say the same about silicates such as talc, kaolin and others.

We recognize that each of these dusts acts in a specific manner upon the pulmonary tissue. We have also learned that a combination of one or more dusts or the action of silica in combination with a non-siliceous dust may alter the pathological and clinical picture. We still do not know too much about these dust diseases. Consequently I believe we should abandon any attempt or habit of referring to these diseases by a generic term unless the specific etiologic agent is mentioned also.

The word commonly in use today is pneumoconiosis. This word means nothing. The proper term is pneumonoconiosis which means something. There

Important →

? look up this term - when first used - is there support for it
G/H

is a tendency to include under this generic term various inhalation diseases of unrelated nature. These occupational dust diseases of the lung comprise a perfectly distinct clinical entity, at least to those who have worked in this field and who are familiar with working conditions and their effects upon health. Metal fume fever, zinc chills, allergic asthma, carbon monoxide poisoning, lead poisoning, mercury poisoning, nitrous fume poisoning ^{are} all inhalation diseases. Certainly there is nothing to be gained by attempting to lump all these under some generic term. I have not mentioned coal. The role of coal dust in causing pulmonary disease is still not clear particularly when we appreciate that many coal dusts contain varying amounts of silica. It is evident that there is an occupational disease of coal miners which by its nature and clinical manifestations can properly be classed as a pneumoconiosis. Much research work is being carried on in many places. Until we know more, it would be an advantage if we abandoned such terms as pneumoconiosis or that still more objectionable term "silicosis" and "siderosis" and use specific definitive terms, viz., occupational dust disease of the lungs - or pulmonary fibrosis - due to the inhalation of silica or of asbestos or ^{of} coal in the so and so industry in such a place.

The terms silicosis and asbestosis have become so widely accepted that it is evident that they will continue to be generally used. The definition of silicosis is not entirely satisfactory, but it has been recognized and accepted by compensation tribunals all over this country. Consequently, I believe that the term silicosis should be restricted to the specific pulmonary disease caused by ^{crystalline} ~~crystalline~~ silica.

Comment

DR. VORWALD: In summary, it would appear that Dr. Lanza is not inclined to retain the word pneumoconiosis but would keep the well-established terms silicosis and asbestosis, which refer to well-defined disease entities with known specific etiology. He deplores, however, the use of terms to identify those pulmonary conditions, caused by inhaled dust, which are not clearly defined with respect to etiology or to clinical signs and symptoms. Those conditions he would refer to as occupational dust disease of the lung due to the inhalation of a specific agent in such and such an industry in such and such a place.

PART ONE

Definitions and Terminology

Chapter ~~One~~ ¹

Definition and Clarification of Terms Pertaining to Pneumoconiosis

A panel discussion dealing with the definition and clarification of terms pertaining to pneumoconiosis was conducted by a panel which had the following members:

Moderator : Arthur J. Vorwald, M.D.

Panel members : Anthony J. Lanza, M.D.

Leonard Greenburg, M.D.

^{senior}
O. A. Sander, M.D.

Richard Wagner

Charles M. Fletcher, M.D.

Discussion, led by A. J. Orenstein, M.D.

^{ion}
Introductory remarks by Arthur J. Vorwald, M.D.

^{The panel}
In planning the agenda for the ~~symposium~~ we were urged by many colleagues to include for discussion the definition and clarification of terms which pertain to the inhalation of atmospheric substances and which have caused or are causing considerable debate. Those colleagues apparently are somewhat disturbed by the lack of uniformity in the use and definition of certain terms, thereby permitting a multitude of interpretations that are often unorthodox and confusing. Although there is some doubt that discussion at this symposium, involving a large group of individuals, would

X Panel

resolve many of the issues concerning the definition, usage, and interpretation of terms, it was felt that the topic should be placed on the agenda if for no other reason than to pave the way for further consideration.

It is true that our discussion concerning definitions and terminology will deal with words and may perhaps be viewed as being too academic or of little practical value. But words are tools and we should try to select the most appropriate tools for our job, a job which pertains to the inhalation of industrial substances and includes diverse interests such as engineering, medicine, law, insurance, compensation and sociology. Our discussion will direct attention to such words as pneumoconiosis, active dust, and inactive dust, and to the terminology for specific disease entities and will attempt to approach the subject from both the medical and the legal point of view. In exploring these two points of view ^{let us} we must consider not only the specialist in diseases of the chest but also the lawyer, the compensation commissioner, the general practitioner, the layman and the patient. The particular interest of each of these individuals should be given consideration in our discussions.

Remarks by Panel member Anthony J. Lanza, M.D.

As a preliminary.....
.....
.....
.....
.....crystalline silica.

from the standpoint of the lay person, the general practitioner, the specialist in diseases of the chest, particularly those concerned with pneumoconiosis, legal counsel, and also the point of view of compensation commissioners.

With that then, the first member of the panel to participate in this discussion is Doctor Lanza, whom you all know and who needs no introduction at this Symposium.

BY DOCTOR LANZA:

Doctor Vorwald, Ladies and Gentlemen: You know, when a preacher gets up to preach or when the politician gets up to orate, particularly if he is looking for votes, they try to create an atmosphere of peace and good will and get everybody in a kind and amiable mood toward themselves, sweetness and light. Such is not my motive. I think that a certain amount of disagreement is healthy and the exploration of disagreements is healthy.

We're concerned with definitions. I often tell my students, when I have any, that if they really want an exercise in mental gymnastics, take a pad and a pencil and try to write a definition, a definition of anything. It's a very difficult thing to do, and they will finish up sadder and wiser.

Now, I think the first point to establish today, if we can establish it, is to try and determine what it is

from the standpoint of the lay person, the general practitioner, the specialist in diseases of the chest, particularly those concerned with pneumoconiosis, legal counsel, and also the point of view of compensation commissioners.

with that then, the first member of the panel to participate in this discussion is doctor lanza, whom you all know and who needs no introduction at this Symposium.

we're talking about. If we can get that far in agreement, maybe we can pull out some gems of light from the discussion.

Now, it seems to me, at least from my point of view, and perhaps I am prejudiced somewhat by my many years of connection with this institution here, that we're concerned with occupational diseases of the lungs due to the inhalation of dust, occupational diseases which occur all over the world, which may cause disability and death, and which do not occur except as occupational diseases.

A man can get lead poisoning by using the wrong kind of hair tonic. He can get carbon monoxide poisoning by being careless in his garage. I never heard of a man getting silicosis working in his garden, or asbestosis from painting up in the garrett. The importance of these diseases has become recognized as far as modern industrial conditions are concerned, within the past forty-five years. The particular disease which has especially attracted attention is the one that we now call silicosis.

About 1918, Doctor Landish of the Phipps Institute who was one of the original group that studied industrial diseases in this country, stated that organic dusts, textile dust, vegetable dust, wood dust, did not cause the definite occupational lung disease which was becoming more and more recognized as the evidence of surveys in many

industries began to accumulate, and revealed that various diseases to which trade names had become attached, were one and the same.

Now, the villain of this piece is silica, principally free silica, and to a somewhat lesser extent, various dusts of combined silica. Now again, it's a good idea to get some background in this business, that is to sufficiently appreciate it.

Everybody breathes silica in one form or another during his lifetime. It composes a major portion of the earth's crust, and it doesn't make any difference whether you walk down the city street or whether you walk across the Nevada desert, you're going to breathe some silica, just the same way that everybody inhales water vapor. The one does not develop dust diseases of the lungs; the other does not drown, but when the dosage of either dust or water becomes too large for the respiratory system to cope with, unfortunate results follow.

So, to repeat, we are concerned with occupational diseases of the lungs caused by the inhalation of dust. These diseases are of slow onset and are characterized by the formation of pulmonary fibrosis, generally accompanied by a varying degree of phthismia. Some of these diseases are further characterized by an increased susceptibility to infection, particularly by the tubercle bacillus. They

present a radiologic picture which may be quite characteristic, but which at times, as all of us have learned to our sorrow, may be quite confusing.

However, these X-ray films have - of dust diseases of the lungs, show a pathological involvement more or less evenly distributed throughout both lungs. The added factor of infection, when present, may confuse the diagnosis, especially when the nature and extent of the dust disease itself is not known. I give you that little background, because it has a certain bearing on what is to follow.

We know a great deal about the occupational disease of the lungs due to the inhalation of crystalline silica. There is, however, still a great deal that we don't know about silicosis, the term commonly applied to this disease, in spite of the fact that a tremendous amount of research has been carried on all over the world, notably in South Africa.

We know less about the action of asbestos, and we know still less about the results of the inhalation of diathermaceous earth. We may say the same about silicates, such as talc, and some of the others. We recognize that each of these dusts acts in a specific manner upon the pulmonary tissue. We have learned that a combination of one or more dusts, or the action of silica in combination

with a non-silicious dust, may alter the pathological and clinical picture. We still do not know too much about these dust diseases.

Consequently, I believe we should abandon any attempt or habit of referring to these diseases by a generic term, unless a specific etiological agent is mentioned also. The word commonly in use today as a catch-all for these dust diseases is the term 'pneumoconiosis'. It is an unsatisfactory term; it's a sloppy term, and it means little or nothing. The proper term is 'pneumonoconiosis, as the term was first used by Zenker. It means something.

However, the term 'pneumoconiosis' has become so widely adopted that it is probably here to stay, so I shall not try to make any battle upon the retention or the abandonment of the term 'pneumoconiosis', for, as I say, I think it's a sloppy term.

There is a tendency to include, under this generic term, various inhalation diseases of unrelated nature. The true occupational dust diseases of the lungs comprise a perfectly distinct clinical entity, at least to those of us who have worked in this field and who are familiar with working conditions and the effects upon health. Metal fume fever, zinc chills, allergic asthma, carbon monoxide poisoning, lead poisoning, mercury poisoning, nitrous fume

poisoning, are all inhalation diseases. The peculiar symptom complex that effects a fair number of people if they come too near a horse or dog or cat or a pig, is familiar to all of you, but there is nothing to be gained by attempting to lump all these diseases under the same generic term, unless you use the term 'lung disease' the way some people use the term 'heart disease', which means anything or nothing, depending upon how you qualify it.

I have not mentioned coal. The role of coal dust in causing pulmonary disease is still not entirely clear, particularly when we appreciate that many coal dusts contain various amounts of free silica. It is evident that there is an occupational disease of coal miners which, by its nature and its clinical manifestations, can properly be classes as a pneumoconiosis. Much research work is still being carried on in many places. Until we know more, it would be an advantage if we could abandon such terms as 'pneumoconiosis' or the still more objectionable term of 'silicatosiis' or 'siderosis', and use specific definitive terms, namely, occupational dust disease of the lungs or pulmonary fibrosis, if you prefer, due to the inhalation of silica or asbestos or coal, in such and such an industry, in such and such a place.

It assumes that any of these agents acts in the same way in all the various places that it's met with on the

face of the globe, and that is highly fallacious, and that is the reason why we get, on the one hand, a tendency to be overly dogmatic, and on the other, a difficulty in reconciling the clinical picture of some of these dust diseases as seen in different parts of the same country or in different countries.

If you wish to give a description of pneumoconiosis to coal miners, for instance, you should specify, 'I am talking about anthracite miners in Pennsylvania; I'm not talking about soft coal miners in Utah or spot coal miners in Alabama'. The absence of clarification of that kind is one of the reasons why there is so much confusion in the general picture of what these dust diseases are all about.

The terms 'silicosis' and 'asbestosis' have become so widely accepted that it is evident that they will continue to be generally used. The definition of 'silicosis' is not entirely satisfactory, but it has been recognized and accepted by compensation tribunals all over the United States. Consequently, I believe that the term 'silicosis' should be restricted to pulmonary disease caused by crystalline silica.

Now, here again, the amount of sloppy terminology and sloppy diagnosis which I see every day, is not only troublesome, but it's maddening. For instance, I got a

report the other day from a physician who had worked up a case of asbestosis and he had done a very good job; the clinical picture, the X-ray, everything was fine. He had worked up his case in first class shape. There was a man exposed to asbestos, and there was no argument as to what was the matter with him, but on his diagnosis for the Compensation Commission, he put third state silicosis.

Now, it seems incredible to me that an intelligent physician could make that kind of mistake, and he wouldn't make that kind of a mistake if he'd ever taken the trouble to find out what this is all about.

Very well, finally, I hope that we can agree that instead of being too concerned about so many of these terms, that we will always mention the etiological agent and, if possible, its chemical composition, or the composition as identified by the X-ray defraction method. Then, anybody who reads your paper knows what you are talking about. Otherwise, he assumes that he knows what you're talking about, and he may be wrong, and the other thing that I hope that we can all agree on is that nobody, capital NO, nobody, will continue to write in this field and invent his own terms to describe what he thinks he sees.

Thank you.

(Applause).

BY DOCTOR VORWALD:

Forgot the bell, not that I want to stop Doctor

Lanza.

It would seem then, if I may summarize just a bit, the points, that Doctor Lanza is inclined to accept the word 'pneumoconiosis', in view of its past history of usage, and perhaps also to accept the well-established disease entity such as silicosis and as bestosis, but he does deplore the usage of other terms and suggests that we agree on identifying these conditions, or precisely that it is pulmonary dust disease, if you will, in such and such a worker in such and such a place, as exposed to such and such material.

The next member of the panel is Doctor Leonard Greenburgh, who is Director of the Division of Industrial Hygiene and Safety Standards of New York State Department of Labor, New York City. Doctor Greenburg too, you know, as well as many of us here do.

BY DOCTOR GREENBURG:

Mr. Chairman, Ladies and Gentlemen: Doctor Lanza's brief discussion makes it possible for me to skip some introductory matter and get down to what I think is a series of answers, or an answer to a series of questions propounded by Doctor Vorwald and sent to each of the panel members.

I think it may be well if we make a little

BY DOCTOR GREENBURG:

Mr. Chairman, Ladies and Gentlemen: Doctor Lanza's brief discussion makes it possible for me to skip some introductory matter and get down to what I think is a series of answers, or an answer to a series of questions propounded by Doctor Vorwald and sent to each of the panel members.

I think it may be well if we make a little

diagram on the board here which is simply indicative, or simply put here to indicate no firm classification of diseases or the course of disease in industrial relations, but merely something suggestive so that we will have something to talk about in trying to define our terms, and I'll read this to you after in case you can't see it from where you are.

I put on the board here simply in outline, a series of squares with various letters in them. We have D in the first, and then -- by the way, I'm talking now about living people, I'm not talking about pathological specimens or laboratory material. 'D' is the man going in industry and gets dust in his lungs. Later on, he may get dust, dust with fibrosis and later on there may be some nodulation present and later on, perhaps, some infection and finally, some dust plus fibrosis, plus nodulation, plus infection, plus conglomeration.

Now, there are some -- and you will hear from one such representative in a few minutes, I presume -- who think that this area here, when the man has dust in his lungs, is a real pneumoconiosis, because in the opinion of these people who are important people in the field, the word 'pneumoconiosis' strictly defines or means dust in the lung or dusty lung. There are others, such as Doctor Lanza, and I may include myself, who believe that this whole

area here, including all of the various stages, is representative of pneumoconiosis and I agree with the British that it's perfectly all right to use the term 'pneumoconiosis' as an abbreviation of pneumoconiosis, and I hardly think it's worth while making an issue of the abbreviation.

At any rate, I would suggest that to my way of thinking, this whole area is a field of pneumoconiosis, and these things may or may not be present. They may or may not be discernible. In fact, in many cases, they are discernible and in many cases not, depending on the particular type of dust.

Now, the second point which I would like to make is, does this happen with organic dust as well as inorganic dust, and mind you, at the beginning, I said that we are not talking about any specific type of pathology. I want all the various types together in this picture, realizing that the coal pneumoconiosis and fibrosis is a different one from the asbestos and from silica.

Well, now that we know, or I think it's pretty well established that coal dust by itself, will produce a pneumoconiosis, and coal dust being an organic dust, I think we should leave the door open to future developments of knowledge and say that this can happen with inorganic dusts and it may also happen with organic. I think one

important difference in the organic and inorganic is probably due to the fact that the inorganic dusts are usually in such large particle sizes that comparatively a small percentage gets up into the lungs, but in the case of coal dust, that's not true and I think it's fairly well established. Our British colleagues will tell us more definitely in a day or two, that coal dust will produce a definite pneumoconiosis, and I would like, however, as this discussion comes out, to say that I think that so far as I'm concerned, I would prefer to see the use of the term 'pneumoconiosis' continued in the literature.

Now, what I say has no -- I haven't said anything about the legal implications of this and the legal implications are perhaps many and perhaps very important. They will have to be discussed by a lawyer. I don't claim to be versed in legal matters, but whatever the lawyer said, I think it's important, perhaps it's fundamentally important for the medical and specialist group in this field, to decide on terminology and then feed that over to the compensation commissions and state legislatures and other bodies who are trying to set up the organic laws dealing with compensation affairs.

So, I would say, in answer to the first question of our Chairman, that pneumoconiosis is a generic term meaning the inhalation of any and all kinds of dust,

organic and inorganic, and having no particular reference to the clinical manifestations. In the early stages, it's impossible to tell and it's important to note, now and forever, that the X-ray is a rather weak tool that we have to rely on a great deal. We have done a great deal of talking about the meaning of the X-ray and, as time goes on, we realize, more and more, at least I feel that we realize more and more, that the X-ray is quite inadequate in many ways.

It's the best we have, but it is inadequate. For example, when you look at the chest picture of a man who has been in a dusty atmosphere, even silica dust, it's impossible to tell the difference in the early stages between these two, particularly when the fibrosis is small in amount, and someone can easily confuse the X-ray picture in the case of an iron miner and be unable to tell whether he is looking at, rather, what some of our colleagues call a benign pneumoconiosis as compared with a real fibrous - early stage of a real fibrosis.

Our Chairman also raised the question, should this term be restricted to those conditions which are accompanied by definite pathology with X-ray and clinical manifestations and with associated pulmonary disability, and which are thus eligible for compensation. That brings up the question as to what you're going to call these

diseases after they're found, and I agree in large part with what Doctor Lanza said, but I agree more thoroughly with the British, and I have a couple of references there which I can read to you if it becomes necessary.

I think what we might call this - these diseases are pneumoconiosis, of say, coal miners, or in other words, silica workers, and then, with or without infection and with or without disability.

Now, it seems to me that if you want to write a compensation act and define your terms fairly accurately, you could proceed on some such basis as this, and you could do a fairly accurate job, if necessary adding some more modifying terms to really pinpoint the type of people whom you wanted to pay disability to. If you wanted to pay them disability with - if you wanted to pay compensation for disability, you would insist on this being present, as is done in New York State at this time.

The next question which our Chairman raised is the question as to whether there are active and inactive dusts as in, I think, -- and I think the answer to that is quite clear. There probably are active and inactive dusts, but the terms are very bad, because we don't know too much about some of these dusts and certainly we don't know too much about how much is present in any one type of exposure.

It's pretty difficult sometimes to tell when you get a man's X-ray picture in the office and you talk to him about his occupation, it's pretty difficult to tell just precisely what he was exposed to and, for that reason, I would think that it would be poor practice to bring in the question of inactive and active dusts.

Furthermore, we have learned in the last fifteen years, that what, at one time, we considered inactive, we now know to be active.

Our Chairman also raised the question as to whether simple silicosis was a condition or a disease. Well, so far as I'm aware, simple silicosis is a disease. It's fibrosis with a certain amount of nodulation, just how much it's impossible to say. I went back to the 1930 report of the Johannesburg Conference, and I would like to read you the definition first used of silicosis, by Doctor Urban to his colleagues. He says: 'If one employs a term simple silicosis to designate a condition of simple silicosis unaccompanied by simple or undetected tuberculosis, one may say that due to a tactical standpoint upon respiratory ailment, the degree of simple silicosis coincides with the appearance of a certain amount of palpable nodulation under the pleura and in the lung substance'.

Apparently, the question as to how much nodulation is somewhat indefinite, and then a little later on,

it's pointed out that, although Urban and his colleagues say that simple silicosis is usually not accompanied by infection, they quote Maragliano's words in which Maragliano says that simple silicosis, even in its earliest detective stage, has an element of low-grade and latent tubercular infection.

I don't want to belabor the point except to say that this is an example of how fallacious the reading of X-ray pictures may be, and it's perfectly possible to read, to further pursue nodulation, than an X-ray picture, and that the autopsy tables find it, and that it's perfectly possible that we do not know where infection takes place.

It used to be set up here at Saranac primarily, and other places, I suppose, too, that when the nodules began to get fuzzy around the edges was when infection began to set in. Well, I think that's pretty indefinite and I think we can not tell from the X-ray picture exactly when infection is and is not present.

Is there a silicosis which antedates nodulation and which is diagnosable? I think the answer to that is definitely yes. I think that the silicosis which has been produced by very finely divided silica, produces a disease which antedates nodulation. In fact, nodulation is very late in that type of disease, and disability does take place.

Then, he has raised the question, shall we use such terms as anthraco-silicosis and sidero-silicosis, and I say I think definitely not. I think that they represent an effort to give a name to something about which we're quite ignorant. We don't know just what the amount of silica is and the amount of coal dust is, in anthraco-silicosis, and I don't think the adding of a name adds anything to it at all.

I think, if the man is a coal miner, it is better to say we have pneumoconiosis in a coal miner, with infection or without infection, with disability or without disability, and I certainly do not prefer to use a new term for every pulmonary condition associated with a special dust. I think it's better to use the name of the dust if you want and the type or description of the type of work, as suggested before, and I agree completely with Doctor Lanza in that regard.

And, finally, he has raised the question as to whether beryllium is or is not in pneumoconiosis. I don't think it's worth while getting into a detailed discussion of that at this time. It perhaps would come better later in the Symposium. Beryllium is apparently primarily a disease of the lung, as we know it, when the patient has inhaled beryllium. The pathology is practically completely limited to the lung. It may be argued that this is not an

irritative process, somewhat similar to silica, let us say. Well, maybe it is and maybe it isn't. I don't know, but I remember the days, in the early days of these discussions, when we thought that the harmful effect of breathing silica dust was due to its sharpness and hardness, and then a short time later, we found out that when workers breathed carborundum dust which was very sharp and very hard, we didn't get the same results as we got in the case of silica dust exposure.

At that time, we gave up the notion that the irritative effects were due to the hardness and sharpness of the dust and it was generally agreed that there must be some peculiar chemical reaction between the tissues and the crystalline silica. Well, if that's true and I presume it is true, -- the aluminum workers would seem to indicate that also -- if that's true, then isn't that somewhat similar to the effect of the entry of silica, of beryllium dust on the tissues of the lungs.

In that respect, beryllium may act very much like silica, and I think it would be unwise at this time to strike out the possibility that beryllium should be classed as a pneumoconiosis.

Well, I don't know that I've added an awful lot to the discussion, but it's one man's view and I think when it's all added together with Doctor Sander and the other

members of the panel, maybe we will be able to come to some agreement on terminology.

Thank you.

BY DOCTOR VORWALD:

Thank you, Doctor Greenburg. Again, Doctor Greenburg does favor the usage of the identifying of dust disease as dust disease in a specific worker. I should like, at this point, Doctor Greenburg, to ask one question, which I'm not clear on at the moment. Do you or do not you favor the use of the term 'pneumoconiosis' as a generic term or do you wish it to be restricted only to those dusts which do produce pulmonary damage?

BY DOCTOR GREENBURG:

No, I would say - I tried to indicate that I favor the larger square in which the term is used for all dust inhalation.

BY DOCTOR VORWALD:

As a generic term?

BY DOCTOR GREENBURG:

Yes, as a generic term; I thought I made that very clear.

members of the panel, maybe we will be able to come to some agreement on terminology.

Thank you.

BY DOCTOR VORWALD:

I don't know if I'm correct, but do I understand that Doctor Lanza favors restricting pneumoconiosis to the dust disease, or do you prefer it as a generic definition?

BY DOCTOR LANZA:

The dictionary definition says, a pulmonary disease characterized by fibrosis, so that would seem to shut out the allergies and a number of other things called pulmonary disease, from inhaling. I think we're dealing specifically with the organic dusts that produce fibrosis.

BY DOCTOR VORWALD:

Do you believe that pneumoconiosis should refer to all dusts or specific dusts?

BY DOCTOR LANZA:

To inorganic dusts.

BY DOCTOR VORWALD:

To all inorganic dusts?

BY DOCTOR LANZA:

If they produce the result; a lot of inorganic dusts don't do anything at all. You're talking about a

O. A. SANDER, M. D.

710 NORTH PLANKINTON AVE.

MILWAUKEE 3, WISCONSIN

September 10, 1952

Arthur J. Vorwald, M.D.
The Trudeau Foundation
P.O. Box 551
Saranac Lake, N.Y.

Dear Art:

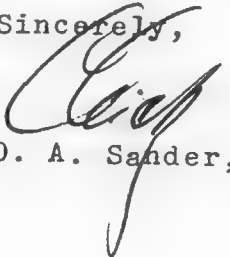
Attached is a first draft of my opening statement for the panel discussion on Monday morning. I am sure that there will be considerable duplication and lack of coordination between the statements. For that reason, most of the statements probably will need much revision before publication in the Proceedings. I'm sure mine will.

As you will note, I'm sticking my neck way out with many positive statements which may be quite unpopular. They certainly ought to start some good arguments. I doubt the need for planted questions, but find one right away which I didn't touch in my statement: "Should combined terms such as anthracosilicosis or siderosilicosis be retained or dropped?" I favor their retention and believe very strongly that "siderosilicosis" should always be used in iron miners with silicosis. I say this because I am sure that the hematite contributes to the roentgen shadows just as with welders with silicosis in foundry cleaning rooms.

Thanks to both Madge and you for the invitations to lunch on Monday and Tuesday, which I accept with pleasure. I am sorry to have to regret the Saturday noon invitation because I cannot get there until about 5 P.M. that day.

Here's to the best-ever Symposium!

Sincerely,



O. A. Sander, M.D.

OAS FH

Enclos.

THE BENIGN PNEUMOCONIOSES

O. A. Sander, M.D., Milwaukee, Wis.

There has been increasing pressure in some quarters to drop this term which was proposed by Pendergrass ten years ago and also to outlaw the terms "anthracosis," "siderosis," "baritosis," and "stannosis." They argue that this nomenclature implies disease and potential or real disability in too many minds and that deposits of inert particulate matter in the lungs (whether or not visualized by X-ray) should not be dignified by an "-osis" term. This school of thinking, as exemplified by the definition of "pneumonconiosis" dreamed up at the Sydney Conference last year, suggests that "carbon, iron, barium, or tin pigmentation" are more benign designations for these non-disabling material deposits.

I am not of this school and have been most unhappy about this turn of events. In my outline of "The Pneumoconioses" which I have been using for teaching for a number of years, I define "pneumonconiosis" simply as "dust in the lungs." This includes all dusts which are small enough to reach the alveoli and which are retained in the lymphatics after they have been phagocytized. This is the true generic meaning of the term and carries with it no implication of any reaction to the dust. By constant repetition and education of persons dealing with these problems, there is no basis for implication of disability in anyone's mind when this term is used.

In my lectures to medical school students, the most convincing

way I have found to clarify the thinking on the "-oses" is to pass around some of Dr. Gough's colored whole lung sections showing moderate coal dust deposits. I point out particularly the localization of the carbon particles in the lymphatics around the blood vessels, giving the "nodular" effect on the cut section. All residents of highly industrialized cities have similar anthracotic pigmentation, which is a true pneumoconiosis due to carbon deposits. Such pneumoconiosis, however, is not a fibrosis, does not predispose to tuberculosis or other infection, and does not cause impaired lung function or disability. Were such carbon radiopaque as is iron, tin, and barium, most of us would have the pneumoconiosis diagnosis made from our chest films. Since it is not, our chest films are read as showing "healthy lungs." Repeated at frequent intervals to the students is that "pneumoconiosis is not synonymous with fibrosis because these inert deposits of carbon also are a pneumoconiosis."

I also do not favor the suggestion that the term "benign pneumoconiosis" be dropped, even though I never have liked it too well because of the implication that all which is not benign is necessarily malignant. It, nevertheless, serves the very useful purpose of setting apart those pneumoconioses which are known not to cause fibrosis or disability and not to predispose to tuberculosis. Perhaps "inert pneumoconiosis" would be a more precise term, but the "benign" will be hard to drop because it has been used so long. Should there be general agreement that it be dropped, then I suggest "fibrogenic pneumoconiosis" as the most descriptive term for silicosis and asbestosis and "inert pneumoconiosis" for siderosis, stannosis, and baritosis.

This raises the question "Are these inert or inactive dusts truly inert at all times and under all circumstances?" For the pneumoconiosis of the South Wales coal miner, it likely is a matter of degree. Minimal

to moderate deposition of this coal dust appears to result in no structural changes, but heavy deposition seems to cause the focal emphysema described by Drs. Fletcher and Gough. The definition of this pneumoconiosis, therefore, must include these differences in the resulting pathology due to the degree of deposition of coal dust.

For the other inert dusts, such as iron, tin, and barium, I have not seen a single case of so much retention that structural change and impaired lung function resulted. All appear to have remained truly inert pneumoconioses. In the siderosis of welders, for example, the iron deposits in the perivascular lymphatics cause no cellular response at all--not even a foreign body reaction. You will note the complete absence of fibrous tissue in these sections of welders' lung (show slides). For the tin oxide pneumoconiosis (stannosis), both Barták in Czechoslovakia and Hughes in this country have shown the absence of fibrosis in two autopsy cases. Function studies by Stocklen of two tin cases were entirely normal. Both had unusually heavy tin oxide deposits as revealed by their chest films. Pendergrass has reported several barium oxide cases (baritosis) in which there were heavy deposits of barium dust in the lungs without pulmonary symptoms. None of these have come to post-mortem study to my knowledge.

In conclusion, I believe very strongly that there is ample cause to retain the present generic definitions of pneumoconiosis and of benign or inert pneumoconiosis and that the specific terms such as siderosis, stannosis, and baritosis be retained as well. With continual education of medical students and the medical profession and with constant repetition of the phrase "pneumoconiosis is not synonymous with fibrosis," the misunderstandings of the past will gradually fade away.

O. A. SANDER, M.D.
710 NORTH PLANKINTON AVE.
MILWAUKEE 3, WISCONSIN

July 17, 1953

Arthur J. Vorwald, M.D.
The Trudeau Foundation
P.O. Box 551
Saranac Lake, N.Y.

Dear Art:

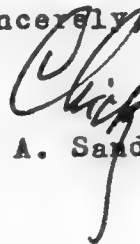
I am sorry for the tardiness of my paper. After getting into it I didn't like any of it, either the submitted manuscript or the stenotypist's notes. I know you will like the revision far better than the original. It follows pretty well what I actually said, but says it better.

Your touch-up of the discussion of that morning session is excellent and I see nothing to change. I hope you will include the letter of February 24 from Miller and Peterson in which they vote to retain the original generic meaning of the term "pneumoconiosis."

I am returning one additional page, No. 33, and hope you can add ".... with the possible exception of category 1." After seeing the sample films, of which I now have a set, I am more than ever convinced that their category 1 is Homer Sampson's old "stage of imagination."

Best regards,

Sincerely,



O. A. Sander, M.D.

OAS FH

Enclos.

Correct By DR Smoker
and
Received
July 17, 1953
PV

Remarks by Panel member O. A. Sauter, M.D.

Dr. Vorwald, Members and Guests of the Symposium:

As defined by Gardner¹, to me "pneumoconiosis" is a general term to designate all types of dust retained in the lungs, with no implication of reaction to the dust or impairment of lung function. There appears to be pressure in some quarters to limit the term to those dust diseases which cause lung fibrosis and potential and real disability. To me, such limitation of the term is as unrealistic as to say that the world now is to be considered flat because it suits someone's convenience.

The reason given to limit the term is quite understandable in some states where the word "pneumoconiosis" is in the compensation law as an occupational disease. The mere diagnosis of pneumoconiosis in those states is sufficient in many cases to constitute liability. Too often there is no attempt to determine the type of dust involved or the extent of the resulting pathological change.

To me the soundest solution of the problem is to consider all of us as having pneumoconiosis because we all are retaining particulate matter in our lungs every day. If we all agree that that is true, then the term has no place in occupational disease laws. Also, the dictionary and textbook definitions must be revised and the "fibrosis" implication removed. There now is much evidence which has been accumulating in the past decade that there are numerous inactive dusts which cause no fibrosis at all.

First of all, let us look at one of Gough's colored gross lung sections showing a moderate amount of coal dust retention, which is

comparable in appearance to most city dwellers' lungs. Note the deposition of the pigment around the blood vessels giving a nodular appearance on cross section of the lung. Were the coal dust radiopaque, the X-ray pattern also would be nodular and the X-ray diagnosis then would be "pneumoconiosis due to carbon dust." But because anthracotic pigmentation in moderate degrees is not visualized by X-ray, such a chest film is read as "healthy lungs," as are most of our chest films. That is precisely why most of the trouble with the term has arisen. Most physicians think of it as a radiological term, whereas it actually means only "dust in the lungs," whether it can be visualized by X-ray or not.

A number of inactive or inert dusts now are recognized which are readily seen radiologically when retained in sufficient amount. First² were the welders' siderosis cases due to retention in the lungs of iron oxide particles. The X-ray pattern is discretely nodular and closely resembles nodular silicosis by X-ray, yet post-mortem studies have shown no evidence of fibrosis. As far as we have been able to determine in the past fifteen years, iron oxide is a truly inert or inactive dust. Grinders and flame cutters of steel may have similar iron oxide exposures of sufficient degree to be visualized by X-ray as nodulation.^{3,4}

The term "siderosis" for such iron pigmentation is a perfectly proper one, but as with pneumoconiosis there must be no implication of fibrosis in the term. Zenker⁵ defined siderosis as a "fibrosis of the lungs due to iron," which is quite understandable because the iron miners' lungs studied by Zenker in 1867 were fibrotic. He incorrectly attributed the fibrosis to the iron rather than to the quartz dust which all the miners had breathed along with the iron. They actually were cases of siderosilicosis.

Additional examples of inert or inactive dusts are tin oxide and barium oxide, which when retained in sufficient degree result in dense sharply defined nodulation by X-ray. Yet in spite of such extreme deposition of particulate material, all studies of such cases have shown a complete absence of fibrosis or impaired lung function. Function studies by ^{Cutter, Faller and ~~and~~ Wilson} Stocklen⁶ of two tin cases were entirely normal. Bartak⁷ in Czechoslovakia and Dundon and Hughes⁸ in this country have shown the absence of fibrosis in detailed post-mortem studies of two cases with tin deposits. The term "stannosis" is perfectly proper for tin pigmentation provided there is no implication of fibrosis in the term. Several barium oxide or baritosis cases have been reported by Pendergrass⁹ in which there were heavy deposits of barium dust in the lungs without symptoms. One of these has come to post-mortem study,¹⁰ which clearly showed the barium deposits, but unfortunately there was an associated anthracosilicosis due to this person's earlier work in anthracite mines.

[unclear]
[unclear]

This brings us to the term "benign pneumoconiosis," first used by Pendergrass¹¹ in 1945, which seems so unpopular in many quarters. My only objection to it is that the benign designation implies that all other pneumoconioses are malignant, which is not true. Some fibrotic pneumoconioses are quite benign, especially when minimal in degree. The term "inert pneumoconioses" probably is preferable. Nevertheless, "benign" most likely will stick because it serves the very useful purpose of setting apart those pneumoconioses which are known not to cause fibrosis or impaired lung function and not to predispose to tuberculosis.

As for the compound terms, I favor the retention of "sidero-silicosis" if both history and X-ray pattern suggest that there has

been sufficient free silica exposure along with the iron to cause silicosis and the root lymph nodes seem definitely enlarged on the chest film. As you know, siderosis alone does not cause enlargement of the root shadows, which makes this an important point in differential diagnosis. "Anthracosilicosis" should be retained only for those cases in which there is a clear-cut history of free silica exposure along with the coal dust and the chest film shows clear-cut evidence of a nodular pattern resembling silicosis. In the absence of significant silica exposure in a coal miner with pin-point or micro-nodulation and emphysema, the term "pneumoconiosis of coal workers" seems the best devised so far. I cannot agree, however, that that diagnosis is proper if the only X-ray evidence of pathological change is emphysema. More about this will be heard from Drs. Fletcher and Hugh-Jones.

In conclusion, I believe very strongly that there are sufficient sound reasons to retain the present generic definitions of pneumoconiosis and the more specific terms of silicosis, siderosis and the others as well. If the general word "pneumoconiosis" is causing so much trouble in medicolegal circles, let's take it out of our occupational disease laws. It should never have been there in the first place. There those pneumoconioses which are known to cause disability when in sufficient degree of development should be so clearly defined that there is no cause for quibbling over terms.

With continual education of medical students and the medical profession and with constant repetition of the statement that "pneumoconiosis is not synonymous with fibrosis," the misunderstandings of the past will gradually fade away. And never forget to add that "you and I--all of us--have a pneumoconiosis."

References

1. Gardner, L. U. The Pathology and Roentgenographic Manifestations of Pneumoconiosis. J.A.M.A. 114: 535-545 (Feb. 17) 1940.
2. Enzer, M. and Sander, O. A.: Chronic Lung Changes in Electric Arc Welders. J. Indust. Hyg. & Toxicol. 20: 333-350 (May) 1938.
3. Sander, O. A.: Benign Pneumoconiosis due to Metal Fumes and Dusts. Amer. J. Roentgen. 58: 277-82 (Sept.) 1947.
4. Hamlin, L. E. et al and Vorwald, A. J. et al. Siderosis--A Benign Pneumoconiosis due to Inhalation of Iron Dust. Indust. Med. & Surg. 19: 151-180 (Apr.) 1950.
5. Zenker, F. A. Ueber Staubinhalationskrankheiten der Lungen. Deutsches Arch. f. klin Med. 2: 116, 1867.
6. ~~Stocklen, J. B.: Personal communication.~~
7. Barták, F. Stannosis. Casop. lek. cesk., 87: 915-32. 1948.
8. Dundon, C. C. and Hughes, J. P. Stannic Oxide Pneumoconiosis. Amer. J. Roentgen. 63: 797-812 (June) 1950.
9. Pendergrass, E. P. Roentgen Diagnosis of Pneumoconiosis and Silicosis. Amer. J. Roentgen. 48: 571-594 (Nov.) 1942.
10. Pendergrass, E. P. and Greening, R. R. Arch. Indust. Hyg. and Occup. Med. 7: 44-48 (Jan.) 1953.
11. Pendergrass, E. P. and Leopold, S. S. Benign Pneumoconiosis. 127: 701-705 (Mar. 24) 1945.

6. Cutler, H.C., Faller, W.W., Stocklen, J.B. and Wilson, W.L. Benign Pneumoconiosis in a Tin oxide Recovery Plant. J. Indust. Hyg. & Toxicol. 31: 139-41 May 1949.

By *duick*

Comment

DR. VORWALD: As I interpret Dr. Sander's views, I believe he is in favor of retaining the word pneumoconiosis and using it in its generic sense, and of having it refer only to the inhalation of dust and not to symptomatology or to the character or extent of pathology within the lung. He agrees with

disease.

BY DOCTOR VORWALD:

So you wish to restrict the use of the term 'pneumoconiosis' to a disease?

BY DOCTOR LANZA:

That's right.

BY DOCTOR VORWALD:

Perhaps sometime later on in the morning, someone will define it for us, what is a disease.

BY DOCTOR LANZA:

Well, if you're sick, you know.

BY DOCTOR VORWALD:

As Doctor Lanza says, if you're sick, you know.

Well, our next member of the panel is Doctor Sander, who is associate in Medicine at Marquette University Medical School, and who is Consultant in Industrial Medicine from Milwaukee, Wisconsin. Doctor Sander has his views regarding this subject, which I believe will be somewhat different than those which have already been expressed.

BY DOCTOR SANDER:

Doctor Vorwald, Members of the Symposium: I'm one who has been teaching students for the last fifteen

BY DOCTOR SANDER:

Doctor Vorwald, Members of the Symposium: I'm one who
has been teaching students for the last fifteen

years, that pneumoconiosis is an overall term covering all inhaled dusts. It simply means dust in the lungs, and to me, limiting the term to those dusts which cause disease or cause symptoms, is like saying it suits our convenience now to say that the world is flat. We know Christopher Columbo said the world was round and they thought he was crazy at that time. We finally have accepted that. Well, now, it suits our convenience, maybe because of our legal friends, to say that the world now is flat again. To me, it's just as ridiculous to limit the term 'pneumoconiosis' to those conditions which cause symptoms.

Dust in the lungs -- we all have some pneumoconiosis, every one of us, those who live in the city or live in cities, even the farmers have some pneumoconiosis, because they inhale organic dusts. It's just as simple as that to me. Let's assume everyone who comes to your office has pneumoconiosis. Then, let's be specific after that. What is it due to?

Speaking of dictionary definitions, Doctor Johnston sent me this, and I think it's too good to pass up. I don't believe in dictionaries, and here is the latest definition, found in Blackstone's New Gold Medical Dictionary, the latest edition. Grinder's Asthma is an interstitial pneumonia due to inhalation of fine particles set free in grinding steel, and then it says, See Fibroid Phthisis, and

then you look up fibroid phthisis, on Page 770, and it says: Chronic slowly progressive pulmonary tuberculosis with extensive fibrosis and mild symptoms. That's the latest edition.

The only textbook I know which has defined 'pneumoconiosis' as an overall term, generic term, meaning any dust in the lung is Doctor Johnston's book. Now, you look up any medical textbook today, and it will say pneumoconiosis is fibrosis due to the inhalation of dust. We have the evidence of dusts which cause no fibrosis, and it's perfectly clear evidence.

We have it, first of all, with the iron oxide, and we have it with the tin oxide. We have it with the barium oxide and there are others. We certainly have it with aluminum oxide. Aluminum oxide doesn't show, because it's not as radiopaque, but it's deposited in the same way. We have it with carbon pigmentation, and in carbon pigmentation the pathologist says, there it is, the paro-vascular lymphatics around blood vessels. It looks like nodular silicosis, doesn't it? It's carbon pigmentation. If that carbon were radiopaque as is iron, tin and barium, we all would have nodulation by X-ray, every one of us. Then we'd agree that we all had pneumoconiosis, but because our chest films are read as healthy lungs, we can't believe that we have pneumoconiosis, but we do. We're all carrying

plenty of material in our paro-vascular lymphatics, and it's not doing any harm, not doing any more harm than these little carbon particles are doing in these paro-vascular lymphatics.

There is just a little difference in the coal pigmentation and coal reaction than you get with the simple soot in the atmosphere. Coal, being a hydrocarbon, causes irritation, undoubtedly, in the bronchials and there is an added factor of obstruction, which undoubtedly we'll hear about from our English friends and Doctor Orenstein, but soot in graphite and non-irritating materials, like iron is non-irritating. You can inject it under the skin and it is the same thing as a tattoo mark. There is no fibrosis. There is no keloid. There is no reaction.

I think we might look at these slides. Many of you have seen these welders sections, but I thought it would be quite important to review it again, because it is basic. I hope you can see the absence of any reaction whatever. Three out of four here have seen these, but probably they're worth seeing again.

This is the welder who worked inside of a tank for ten or eleven years. There was no attempt at ventilation so he had heavy welding fume exposure. Welding fume, you know, is ninety-nine percent plus, iron oxide, very fine dust. He had no symptoms. He had this nodulation,

which when we saw the film in 1935, we scratched our heads and said, he probably has silicosis. He has no silica exposure, and we wondered, is there possibly a free silica liberated in the carbon arc from the coating of the welding rod. After all, there is sodium silicate binder in the coating, and we investigated that and there was no evidence of any SiO_2 . Well, this fellow, unfortunately, died a year later, and we were able to get his lungs.

May we see the next, please? And, this is the low part view, showing the deposits of pigment in the parovascular lymphatics. The next is a high power view. Note the absence of fibrosis. Here is a high power view, showing the extreme amount of pigment deposit in the parovascular lymphatics. That pigment is inside the parovasculars. Grossly, that lung looked just like an anthrapigmentation, which you see in most adults living in cities, but with some ferrous cyanide stain, we were able to prove this was iron oxide from his welding fumes. Note the absence of fibrosis, and in the next slide, is the one for connective tissue, showing complete absence of fibrosis, no fibrous tissue reaction, no intelligible virus.

We have the additional evidence from the tin cases. We have had - there have been two post mortem studies, one by Bartuk in Czechoslovakia, and the other by Doctor Hughes and both showed a complete absence of fibrosis in these tin

deposits. Unfortunately, I didn't bring my slides, but you all have seen the extreme pigmentation with the tin oxide, and we have two good post mortem cases, which show a complete absence of fibrosis, and just an inert deposition.

Bartuk did complete function studies on his cases and found no decrease in pulmonary function. The barium cases of Doctor Prendergast have no symptoms of any kind. There are not too many barium cases. There hasn't been that much barium oxide exposure, but there again, we have this marked deposition of inert material, which causes no fibrosis.

Therefore, I say there is such a thing as inactive dust and no matter how much iron oxide, no matter how much tin oxide, no matter how much barium oxide you inhale, you are not going to get symptoms. We have yet to see a case of tin oxide with symptoms. They haven't seen any in Cleveland where they have uncovered a good many. Bartuk had no case with symptoms, in spite of the extreme X-ray evidence of it. So there is such a thing, in my opinion, of inactive dust, and I don't like the term benign pneumoconiosis any more than you do.

I wish Doctor Gene Prendergast were here to defend himself. He coined the term, you know, about ten years ago. Benign implies that the other pneumoconioses are malignant. I don't like it for that reason because

they're not all malignant either.

I think inert pneumoconiosis would be a perfectly proper term for the pure iron oxide, the pure tin oxide, and the pure barium oxide deposits. I think it would be perfectly proper because we have the pathological evidence that it does not cause reaction, that it doesn't cause symptoms.

As for the retention or discard of such terms as anthraco-silicosis or sidero-silicosis, I think it would be perfectly proper to retain sidero-silicosis where you know that there has been both silica and iron exposure. Every iron miner, no matter where he works, who has any exposure to silica, and most of them do have exposure to silica, we have been looking for iron miners who have never had exposure to silica, had only hematite exposure, and we haven't found one so far. Every miner has a sidero-silicosis if he develops anything at all. We know he has hematite in his lungs, and I firmly believe that some day we're going to be able to show that this hematite itself, in itself, is adding to the nodulation which we see by X-ray.

We have seen it in a few post mortems in iron miners, where there was considerable iron pigmentation, which undoubtedly helped produce the nodulation. I have yet to find, however, a miner who had only iron pigmentation, that is that he had a nodular pattern in his X-ray

film where the pattern was due only to iron pigmentation. We haven't found such a case and we may not, because all of the miners in the past, the older men who have nodulation, by X-rays, always helped drill the shafts, and the shafts, as you know, are in silica, in quartz. They're usually quartz veins, so even the mucker, who has only ore dust exposure, has had silica exposure in the past, because he usually helped in the drilling of the shafts.

I believe, firmly believe, that it is perfectly proper to retain the term sidero-silicosis. As for the coal miners pneumoconiosis, I'm definitely in favor of throwing out the term anthraco-silicosis. We know that there is a pathology, in the absence of any significant amount of free silica with coal dust. Doctor Fletcher and his coal workers have shown that very clearly, and you will hear more about that later. I do not believe that that term is proper in the majority of coal miners pneumoconiosis cases.

Now, as for pneumoconiosis itself, I'm in favor of retaining it, and keeping it as an overall term, and always driving home to whomever you're talking to, whenever you're thinking about it, that you and I, all of us have some pneumoconiosis.

Thank you.

BY DOCTOR VORWALD:

Thank you, Doctor Sander. You have heard Doctor Sander express his views, which are somewhat different than those expressed by the previous members of the panel. As I interpret it, Doctor Sander is in favor of retaining the word 'pneumoconiosis' and using it in its generic sense, that is that it has reference only to the inhalation of dust, and does not have reference to either the symptomatology or the character or extent of pathology within the lung.

Furthermore, he agrees with an expression by Doctor Greenburg, that there are active and inactive dusts. I'm sure that we, too, would agree with that view. Doctor Sander is in favor, therefore, of trying to identify some of those conditions as pneumoconioses which are produced by inactive dusts. He is inclined to retain the term sidero-silicosis, but is in favor of discarding, at least for the time being, the term anthraco-silicosis, I believe, in the final analysis, on the basis that we just do not know what the precise etiological agent is which is giving rise to this pulmonary change, as seen, for example, in coal workers.

I should merely like to put a question to the audience at this time for consideration. Since there are active and inactive dusts, and should we restrict the term pneumoconiosis to those pulmonary conditions which are

Association of Casualty and Surety Companies

Symposium

CASUALTY DEPARTMENT

RICHARD C. WAGNER, MANAGER

ANDREW KALMYKOW, ASSISTANT MANAGER

SIXTY JOHN STREET
NEW YORK 38, N.Y.

September 12, 1952

Dr. Arthur J. Vorwald
Director, The Trudeau Foundation
and The Saranac Laboratory
P. O. Box 551
Saranac Lake, New York

Dear Dr. Vorwald:

I am enclosing two copies of my paper to be delivered on
the first day of the symposium. I am sorry I could not get
this to you any sooner.

Very truly yours,

Richard C. Wagner
Richard C. Wagner, Manager
Casualty Department

RCW:HLH
Enc.

DEFINITION OF PNEUMOCONIOSIS - SOME LEGAL ASPECTS

Paper Read By

Richard C. Wagner, Manager, Casualty Department,
Association of Casualty and Surety Companies, at
Seventh Saranac Symposium, on September 22, 1952,
at The Saranac Laboratory, Saranac Lake, New York.

One of
two
Copies -
one in Mosler's

The question of definition of medical terms would seem, as a matter of first impression, to be a problem exclusively for the medical profession. Certainly it can be conceded that it is primarily a medical problem. Obviously that was the view of the sponsors of this meeting, for here I am the only attorney in a group of five eminent physicians. However, somewhat closer study reveals, as is often the case in the field of occupational diseases, that medical definitions have important, often vital, bearing on legal rights both of employers and employees. This is especially true where such definitions, and any defects they may contain, are incorporated into any statute. They are also of importance where they form part of the testimony or influence the judgment of a medical witness.

Let me illustrate. We are here interested in the definition of the term "pneumoconiosis" and those of the other diseases or conditions that fall within the broad scope of that general term. In New Hampshire for example, the Workmen's Compensation Law provides compensation for certain listed diseases, including "silicosis and other pulmonary dust diseases." Whether a particular condition can be classed as a pulmonary dust disease will determine whether or not that particular individual will receive compensation therefor. The ultimate determination in such a case is very likely to rest on the medical testimony which is presented. Does beryllium poisoning, for example, fall within this category? What about an allergic reaction to let us say flour dust?

A medical expert confronted with the necessity of expressing an opinion on the point might be inclined, if my experience is any criterion, to a liberal interpretation in order to enable the claimant to receive compensation. What

would his testimony be, however, where instead of resulting in the granting of compensation, classifying a particular condition as a pulmonary dust disease would result in reducing or possibly barring the payment of compensation. As no doubt many of you know, many states have special provisions with respect to silicosis and some of the other dust diseases. There are provisions for graduated scales of benefits, the so-called escalator clauses, whereby compensation for disability occurring shortly after the effective date of the law is limited. Special requirements as to exposure or time limitations are also provided. The particular facts of a specific case might well not fit those particular requirements. Yet consistency is necessary. A different answer to the same question can hardly be justified because of the difference in the effect such an answer would have. At the same time, it might be well if in formulating the definitions on which opinions have to be based, the legal effect of the use of particular language may well be given some consideration. Their effectiveness as useful tools in the solution of practical problems would thereby be enhanced.

I have indicated that the problem of definitions is of significance under a schedule type law. It is of importance also, particularly with respect to dust diseases, under a law providing broad occupational disease coverage either with or without definition. As I have mentioned, a number of states have found it necessary because of the special nature of the dust diseases, to include provisions specifically dealing with such diseases which differ from those relating to occupational diseases generally. A general descriptive term is necessary to describe the diseases to which these special provisions should be applicable. At the same time, such a term should not be so broad as to be applicable to conditions to which those special provisions should not apply. The term would have to be general for if specific diseases, say silicosis or asbestosis, only are mentioned, avoidance of these special provisions may be attempted by alleging disability from one of the other dust diseases.

The legislative history of such provisions in Michigan is interesting in this connection. When provisions for compensation for occupational diseases were first enacted in 1937, the schedule of such diseases included three different references to dust diseases. One was stone workers or grinders phthisis caused by quarrying, cutting, crushing, grinding or polishing of stone, or grinding or polishing of metal. The other was silicosis caused by mining. The third referred to pneumoconiosis caused by quarrying, cutting, crushing, grinding or polishing of metal. It is apparent that the Legislature in this instance was having some difficulty with definitions. In 1943 the schedule was repealed and a broad definition substituted. In that state the special dust disease provisions both before and after the 1943 amendment referred to "silicosis or other dust disease." From the use of the word silicosis the inference seems plain that the disease must be pulmonary. Yet I believe the special inclusion of that word would have been desirable. Some doubt as to the scope of these provisions, however, must have existed for in 1945 when provisions for periodic physical examinations were added these referred to "silicosis, pneumoconiosis or other dust disease." These, I may add, have since been repealed when because of indefiniteness they were held unconstitutional by the court.

This legislative history is of interest as indicating the difficulty experienced in the use of terms in just one state. It might be noted further that while the schedule was in force in Michigan there was considerable question whether silicosis was compensable in any industry except mining and whether the term pneumoconiosis was broad enough to include silicosis, *Sutter vs. Kalamazoo Stove and Furnace Company*, 1941, 297 Mich. 226, 297 N.W. 475; *Gerlesits vs. Lakey Foundry and Machine Company*, 1947, 319 Mich. 229, 29 N.W. 2d 856; *Mercante vs. Michigan Steel Casting Company*, 1948, 320 Mich. 542, 31 N.W. 2d 712.

While we are discussing terms, I would like to express a word of caution.

If a condition has a name, there is a tendency among laymen to feel that it is disabling and that it should be compensable. Many different terms, and new ones seem to be cropping up with surprising frequency, have been said to come within the general scope of the word pneumoconiosis. If that term or an equivalent is used without adequate definition in a law, any condition which might be said to come within its scope is deemed to be compensable. Yet, if I am not mistaken, a number of these are not considered to be disabling. The Fifth Edition of Gould's Medical Dictionary (1941) defines pneumonokoniosis as:

"A general term applied to chronic induration or fibrous inflammation of the lungs due to the inhalation of dust. Various names are given to it according to the kind of dust causing the inflammation: anthracosis, that due to the inhalation of coal-dust; siderosis, that due to inhalation of metallic dust; chalicosis, that due to the inhalation of mineral dust."

A layman might well feel that all these conditions are or should be compensable. Let us look, however, at the 1949 Edition apparently of the same book now known as "Blakiston's New Gould Medical Dictionary" First Edition. Pneumoconiosis is defined as:

"Chronic inflammation of the lungs caused by the inhalation of dust. All of the recognized forms are due to mineral dusts. (Neither irritations of the bronchial tree nor acute infections resulting from inhalation of organic dusts are classed as pneumoconioses.) The predominant reaction is fibrosis, which varies in type with the etiologic dust. Silicosis and asbestosis are the main forms of pneumoconiosis known to cause disability. Other forms, known as benign pneumoconioses, in which the reaction is limited to the stromal tissues, are anthracosis, due to carbon dust, siderosis, due to iron dust, calcicosis, due to marble dust, and baritosis, due to barium dust. Also see beryllosis."

I think you will agree that there is considerable difference in the effect these two definitions would have on laymen.

That my point is not academic, is evidenced by a law recently adopted in Alabama. This law enacted in 1951 (Act No. 180, Laws of 1951, effective June 29, 1951) provides compensation only for "occupational pneumoconiosis." This is defined as:

"A disease of the lungs caused by inhalation of minute particles of dust over a period of time and which said dust is due to causes and conditions arising out of and in the course of the employment without regard to whether or not said causes or conditions are inherent in the employment or can be eliminated or reduced by due care on the part of the employer. The term 'occupational pneumoconiosis' shall include, but without limitation, such diseases as silicosis, siderosis, anthracosis, anthra-silicosis, anthraco-silicosis, anthraco-tuberculosis, tuberculo-silicosis, silico-tuberculosis, aluminosis, and other diseases of the lungs resulting from causes enumerated in this section."

Please note the inclusion of siderosis, anthracosis and aluminosis. I would dislike to attempt to define with any degree of certainty the scope of this law.

Yet at least some of you may be called upon to give an opinion whether or not a specific condition is or is not "occupational pneumoconiosis" within the meaning of this definition.

Some of the other statutory references to dust diseases may be of interest. In New York the reference is to "silicosis or other dust diseases." A rather interesting situation arose under this provision. In 1942 an employee claimed and was awarded compensation under Article 4-A, since repealed, relating to dust diseases. The claim was for disability alleged to have been suffered from exposure to dust from vacuum cleaners, *Sturesky vs. Straussman*, 1941, 263 A.D. 771, 30 N.Y.S. 886. After a period in military service he reopened his case. He now contended

that his condition, asthma, was not a dust disease but an "occupational disease." The court held that its original decision was not res adjudicata and on the basis of the medical evidence presented, affirmed an award under the broad occupational disease provisions. Apparently, when the original claim was presented, there was doubt whether the claimant's condition was an occupational disease or an ordinary disease of life. The contention that it was a dust disease probably was thought to strengthen his case. Once the occupational nature of his condition was established, apparently to avoid the limiting provisions of Article 4-A, he was able successfully to reject this theory (id. 1948, 273 A.D. 1036, 78 N.Y.S. 2d 633). I may add that in Missouri, on the other hand, asthma from an allergy to wheat dust was held not to be an occupational disease since it was due to the claimant's sensitivity and not to the hazards of the employment as such, Sanford vs. Valier-Spies Milling Co., 1950, 235 S.W. 2d 92.

In Ohio the silicosis provisions are also applicable "to other occupational diseases of the respiratory tract." This language seems somewhat broader than the reference to dust diseases or pulmonary dust diseases found in some of the other laws. It may, for example, well result in a different construction than that which was reached in New York. In Ohio it is interesting to note that berylliosis is specifically excluded from these provisions although the medical experts known as silicosis referees are specifically given jurisdiction with respect to this disease.

A law providing compensation for occupational diseases was enacted in Louisiana this year. This contains a list of compensable diseases, including asbestosis, silicosis and pneumoconiosis. In view of the fact that the courts administer the Workmen's Compensation Law in that state, the interpretation of this term may well present some problems, in the solution of which doctors will have to play an important part.

Besides the definition in Alabama, Florida comes nearest to having a

definition of dust diseases in the statute itself. In that state, compensation is provided for occupational diseases under a broad definition. It is there further specifically stated that, "disability from silicosis, asbestosis or any dust disease shall be caused only from the characteristic fibrotic condition of the lungs caused from the inhalation of dust." Thus here we find with reference to dust diseases a specific reference to fibrosis which you will have noted is lacking in some of the other statutory references.

There are, of course, a number of states that make specific mention, either with or without definition, of silicosis and asbestosis. These definitions are fairly similar in wording although they do differ in exact language from state to state. Some include a specific reference to nodulation with respect to silicosis. Others refer to the "characteristic fibrotic condition of the lungs caused by inhalation" of silicon dioxide dust or asbestos dust, as the case may be. I have not heard of any particular difficulty arising from these definitions. Some thought might be given, however, to whether or not the reference to nodulation should or should not be made.

I have endeavored to point to some of the more important legal problems that arise through the use of terms without definitions or where the definitions used are not completely satisfactory. In illustration, specific reference to the provisions of the laws of the various states and experience thereunder has been made. While these are legal problems, lawyers of necessity must look to the medical profession for their solution. I believe it would be of mutual benefit if in formulating such terms and their definitions the medical profession kept some of these questions in mind. Is there a general descriptive term that would cover silicosis and other similar dust diseases and, at the same time, exclude non-disabling conditions? What is and what should be covered under the term pneumoconiosis? These and other questions will require an answer. It would be most helpful if such answers were based on informed opinion reached after mature deliberation.

DEFINITION OF PNEUMOCONIOSIS - SOME LEGAL ASPECTS

Paper Read By

Richard C. Wagner, Manager, Casualty Department,
Association of Casualty and Surety Companies, at
Seventh Saranac Symposium, on September 22, 1952,
at The Saranac Laboratory, Saranac Lake, New York.

The question of definition of medical terms would seem, as a matter of first impression, to be a problem exclusively for the medical profession. Certainly it can be conceded that it is primarily a medical problem. Obviously that was the view of the sponsors of this meeting, for here I am the only attorney in a group of five eminent physicians. However, somewhat closer study reveals, as is often the case in the field of occupational diseases, that medical definitions have important, often vital, bearing on legal rights both of employers and employees. This is especially true where such definitions, and any defects they may contain, are incorporated into any statute. They are also of importance where they form part of the testimony or influence the judgment of a medical witness.

Let me illustrate. We are here interested in the definition of the term "pneumoconiosis" and those of the other diseases or conditions that fall within the broad scope of that general term. In New Hampshire for example, the Workmen's Compensation Law provides compensation for certain listed diseases, including "silicosis and other pulmonary dust diseases." Whether a particular condition can be classed as a pulmonary dust disease will determine whether or not that particular individual will receive compensation therefor. The ultimate determination in such a case is very likely to rest on the medical testimony which is presented. Does beryllium poisoning, for example, fall within this category? What about an allergic reaction to, let us say, flour dust?

A medical expert confronted with the necessity of expressing an opinion on the point might be inclined, if my experience is any criterion, to a liberal interpretation in order to enable the claimant to receive compensation. What

would his testimony be, however, where instead of resulting in the granting of compensation, classifying a particular condition as a pulmonary dust disease would result in reducing or possibly barring the payment of compensation. As no doubt many of you know, many states have special provisions with respect to silicosis and some of the other dust diseases. There are provisions for graduated scales of benefits, the so-called escalator clauses, whereby compensation for disability occurring shortly after the effective date of the law is limited. Special requirements as to exposure or time limitations are also provided. The particular facts of a specific case might well not fit those particular requirements. Yet consistency is necessary. A different answer to the same question can hardly be justified because of the difference in the effect such an answer would have. At the same time, it might be well if in formulating the definitions on which opinions have to be based, the legal effect of the use of particular language may well be given some consideration. Their effectiveness as useful tools in the solution of practical problems would thereby be enhanced.

I have indicated that the problem of definitions is of significance under a schedule type law. It is of importance also, particularly with respect to dust diseases, under a law providing broad occupational disease coverage either with or without definition. As I have mentioned, a number of states have found it necessary because of the special nature of the dust diseases, to include provisions specifically dealing with such diseases which differ from those relating to occupational diseases generally. A general descriptive term is necessary to describe the diseases to which these special provisions should be applicable. At the same time, such a term should not be so broad as to be applicable to conditions to which those special provisions should not apply. The term would have to be general for if specific diseases, say silicosis or asbestosis, only are mentioned, avoidance of these special provisions may be attempted by alleging disability from one of the other dust diseases.

The legislative history of such provisions in Michigan is interesting in this connection. When provisions for compensation for occupational diseases were first enacted in 1937, the schedule of such diseases included three different references to dust diseases. One was stone workers or grinders phthisis caused by quarrying, cutting, crushing, grinding or polishing of stone, or grinding or polishing of metal. The other was silicosis caused by mining. The third referred to pneumoconiosis caused by quarrying, cutting, crushing, grinding or polishing of metal. It is apparent that the Legislature in this instance was having some difficulty with definitions. In 1943 the schedule was repealed and a broad definition substituted. In that state the special dust disease provisions both before and after the 1943 amendment referred to "silicosis or other dust disease." From the use of the word silicosis the inference seems plain that the disease must be pulmonary. Yet I believe the special inclusion of that word would have been desirable. Some doubt as to the scope of these provisions, however, must have existed for in 1945 when provisions for periodic physical examinations were added these referred to "silicosis, pneumoconiosis or other dust disease." These, I may add, have since been repealed when because of indefiniteness they were held unconstitutional by the court.

This legislative history is of interest as indicating the difficulty experienced in the use of terms in just one state. It might be noted further that while the schedule was in force in Michigan there was considerable question whether silicosis was compensable in any industry except mining and whether the term pneumoconiosis was broad enough to include silicosis, *Sutter vs. Kalamazoo Stove and Furnace Company*, 1941, 297 Mich. 226, 297 N.W. 475; *Gerlesits vs. Lakey Foundry and Machine Company*, 1947, 319 Mich. 229, 29 N.W. 2d 856; *Mercante vs. Michigan Steel Casting Company*, 1948, 320 Mich. 542, 31 N.W. 2d 712.

While we are discussing terms, I would like to express a word of caution.

If a condition has a name, there is a tendency among laymen to feel that it is disabling and that it should be compensable. Many different terms, and new ones seem to be cropping up with surprising frequency, have been said to come within the general scope of the word pneumoconiosis. If that term or an equivalent is used without adequate definition in a law, any condition which might be said to come within its scope is deemed to be compensable. Yet, if I am not mistaken, a number of these are not considered to be disabling. The Fifth Edition of Gould's Medical Dictionary (1941) defines pneumoconiosis as:

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accompanied by signs and symptoms, then what do we do with those dusts which are inactive and give rise to no such symptoms, if we can not refer to them as belonging to the category of pneumoconiosis? Consider that for a bit.

In the introductory remarks of this panel, I mentioned that the terms and definitions have interest to fields of endeavor other than medicine, and so we have asked the next member of our panel, Mr. Wagner, to present his views. Mr. Wagner is Manager of the Casualty Department, of the Association of Casualty and Surety Companies, New York City, New York. Mr. Wagner.

BY MR. WAGNER:

Doctor Vorwald, Ladies and Gentlemen of this Symposium: I, unfortunately, am going to have to stick pretty close to this paper, and I realize that makes a rather dry presentation, but if I start to bandy around some of these medical terms, as a layman, I'm afraid I'm going to get into trouble, so I might be in trouble anyway, but I'll stick to what I have here.

(Mr. Wagner read his prepared paper, copy of which is on file in the Saranac Laboratory).

BY DOCTOR VORWALD:

You have heard the representative from the legal

BY DOCTOR VORWALD:

You have heard the representative from the legal

profession, and I think it is evident that that profession is placing upon the medical group, the task, and pleading for clarification and definitions of terms by the medical profession, for use and application by the legal group. They look upon the medical profession for those definitions and that clarification.

Our next member on the panel comes to us from England. Doctor Fletcher is Director of the Pneumoconiosis Research Unit of the Medical Research Council in Cardiff, Wales, and has had much experience with coal miners in that area, and he is also a lecturer of the Post Graduate School of Medicine, University of London, London, England, and I'm happy to introduce to you Doctor Fletcher.

BY DOCTOR FLETCHER:

Mr. Chairman and Gentlemen: I'm in slight difficulties, since this is the first occasion on which I've attempted to take part in a panel of this kind and I don't know to what extent I'm expected to answer the questions that Doctor Vorwald fired at me across the Atlantic, or to what extent I can throw brickbats at the - some of the remarks which I regard as highly debatable, which have been made already this morning. I will attempt to combine the two.

I have been pleased with the definition of



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15th September, 1952

Dear Art,

I enclose my comments on your question concerning the definition and classification of terms pertaining to pneumoconiosis. I have added an extra paragraph at the end on pneumoconiosis without radiological change, which is a problem upon which I should be glad to have the views of the Panel.

If it is also suitable for the discussion, would the following question be considered by the Panel?:-

"Since radiography provides the single most important piece of evidence in the diagnosis of pneumoconiosis, the present lack of agreement as to the earliest characteristic radiological signs of pneumoconiosis and as to the method of classification of the more advanced stages, is an important source of national and international confusion. Does the Panel agree that if an international body, such as W.H.O. or I.L.O. were to assemble sets of radiographs derived from men working in some of the major pneumoconiosis-risk industries and were to circulate them for classification to leading experts in different countries, some measure of present agreement and disagreement would be obtained and that, in the light of these findings, a conference of the experts concerned might be able to agree upon a common system? Later circulation of further sets of films might then ensure that the system was being used in the same way in different countries."

I am not sure whether this is an appropriate topic for the Panel. I have been trying to stimulate I.L.O. to do something about this question of radiological classification for two years without success, but I think it is possible that W.H.O. might be stimulated by a resolution emerging from a Saranac Symposium.

Yours sincerely,

Charles

*Very much looking forward to my trip.
I will bring the letters & my other papers with me*

Pneumoconiosis

Strictly, pneumoconiosis means a "process in the lungs caused by dust" and is theoretically initiated when the first dust particles become lodged in the lung parenchyma and cause any reaction. For practical purposes the definition of pneumoconiosis proposed by the International Conference of the I.L.O. in Sydney in 1950 is reasonable: "Pneumoconiosis is a diagnosable disease of the lungs produced by the inhalation of dust, the term 'dust' being understood to refer to particulate matter in the solid phase but excluding living organisms". The terms "diagnosable" here implies the presence of signs or symptoms, but not always loss of function (paras. 5 and 27 of the report of the Conference). To me, the word "disease" here implies any abnormal condition of the lungs. It is always difficult to define just where normality ends and abnormality begins. No sharp distinction can be drawn between the accumulation of soot or dust in a town dweller's lung and the earliest changes of coal pneumoconiosis, or even silicosis. (1, 2). It would be reasonable in some cases to speak of "town dwellers' pneumoconiosis". In scientific medicine the diagnosis of pneumoconiosis should thus be made as soon as any characteristic change can be detected, whether this be clinical or radiological during life, or histological after death, so long as this change can be reasonably attributed to the presence of dust. In most forms of pneumoconiosis histological changes are the first to appear, and radiological abnormality precedes the appearance of clinical or functional changes by many years (although in some forms of pneumoconiosis such as asbestosis and byssinosis and, as I shall show later, in some cases of coal pneumoconiosis, the reverse is true). These radiological changes are due to dust in the lung which, for scientific and logical reasons, must be regarded as constituting pneumoconiosis.

The chief objection to applying the term pneumoconiosis to cases without disability is psychological. A man may be told that he has pneumoconiosis on the grounds of radiological evidence, and may then be disgruntled if he be not granted compensation: or, in an area such as South Wales where large numbers of men are seriously affected by the disease, he may be alarmed by the diagnosis and develop an anxiety state. It would, therefore, be advantageous to have a separate term which could be used for non-disabling pneumoconiosis, but since non-disabling and disabling pneumoconiosis are usually but different stages of a single pathological process, it would be illogical in scientific medicine to use separate terms. Moreover, the radiologist or pathologist, as he classifies radiographs or sections of lungs, cannot distinguish the disabled from the non-disabled and must apply the term pneumoconiosis to all those in whom he finds abnormality attributable to dust. We use the term tuberculosis to cover both the healed apical scar and extensive active disease; we distinguish the two by suitable prefixes, and sensible doctors do not alarm their patients by telling them that they have pulmonary tuberculosis when they discover an apical scar in a routine radiograph.

For purposes of compensation it may be necessary to define a limit of acceptable abnormality (whether clinical or radiological) below which an official diagnosis of pneumoconiosis will not be made, above the lowest limit recognised

by special investigators. It might be appropriate to describe pneumoconiosis of severity sufficient to merit compensation as "compensatable pneumoconiosis", and to define its limits for each form of pneumoconiosis in appropriate histological, radiological, clinical or functional terms.

In the Pneumoconiosis Research Unit we have not found it difficult to explain to a man that his radiograph "shows that he has been a miner, but does not show enough dust to do any harm" and that he cannot therefore expect compensation. In border-line cases also we have found no difficulty in explaining that doctors may disagree with one another in assessing the severity of the disease so that in such cases, while application for compensation may be worth making, the man must not be surprised if it fails despite our own diagnosis of pneumoconiosis. We have found the Welsh coal miner much more ready to accept the existence of observer error among doctors than are members of the medical profession themselves!

Active and Inactive Dusts

The pathological activity of a dust depends not only on its chemical nature but also on its physical characteristics, such as size-distribution, and upon the dose administered, so that no simple classification of dusts into active and inactive is possible. For example, a given quantity of 20 Angstrom silica may be highly active if given in a single large dose, but the same quantity may have no effect at all if administered in small concentration over a longer period.

to / It might be possible to reach a theoretically sound classification of dusts according to their activity on the basis of the relationship between the amount of material retained in the lung and the degree of pathological change. Thus, an active dust would be one in which the degree of pathological reaction and its presence in the lung is great in relation to the amount of retained material (e.g. silica and beryllium) and an inactive dust would be one in which the pathological change is relatively small (e.g. coal and iron), but here we meet the difficulty that dusts such as coal which would be classified as inactive per se may, in combination with tuberculous infection, produce gross pathological changes.

A vast amount of study would be required to achieve anything but a rough classification along these lines and it would still be of little practical use. As I hope to show in my paper on the Epidemiology of Coal Miners' Pneumoconiosis, we now have evidence that Welsh anthracite or steam coal dusts produce much more pneumoconiosis in men exposed to them than do English bituminous coal dusts in the same airborne concentration, and yet the degree of fibrosis in the lungs of anthracite and bituminous coal miners in relation to the amount of retained coal does not appear to be very different. These different effects may be due to differences in the proportion of the inhaled dust retained in the lung, the reason for which we do not understand.

Any classification of dusts into active or inactive on the basis of animal experiments may be dangerously misleading. Coal dust was for years regarded as harmless in Great Britain and is still so regarded in Germany, largely because animal lungs do not show any fibrotic response to inhaled coal dust, and yet the

Silicosis

I am unaware of any difference of the meaning of the words "disease" and "condition" in relation to silicosis. I regard any pathological change attributable to free silica as a form of silicosis. I distinguish the formation of uniformly distributed, concentric, collagenous, fibrotic nodules by the term "Classical nodular silicosis".

Terms for Specific Entities

The word silicosis should only be used when it has been clearly demonstrated that the pathological changes in the lung are attributable to the action of silica. So far as I know this can only be done where the dust exposure is to pure silica or where the reaction in the lung is of the classical type. For all other pulmonary disease due to dust the word pneumoconiosis should be used, preceded by the name of the dust or occupation by or in which the disease is caused. The term "anthraco-silicosis" should be applied only to those cases of classical silicosis in which there are also deposits of coal in the lungs (for example, rock workers in coal mines) but should not be applied to the ordinary coal workers' pneumoconiosis in which there is no evidence that silica is responsible for the fibrosis.

Clarity is gained by using prefixes to the general term "pneumoconiosis" for the pulmonary conditions attributable to specific dusts.

Pneumoconiosis Without Radiological Change

In South Wales Professor Gough has occasionally demonstrated cases of severe focal emphysema in simple coal pneumoconiosis which must have been disabling during life but which was unassociated with any radiological abnormality. Dr. Hugh-Jones will show in his paper tomorrow that the impairment of ventilatory function in coal miners with simple pneumoconiosis is not closely related to the severity of the radiographic changes and that coal miners without any radiological abnormality appear, on the average, to have impaired ventilatory capacity when compared with non-miners of the same age. This impairment is, in some cases, associated with a tendency to bronchial spasm. Whether this disability is attributable to dust inhalation we cannot yet say. Further studies of much larger samples of miners and of men from various other dusty and non-dusty occupations will be required to establish its significance.

But these preliminary findings do suggest that we may at present pay too much attention to the radiological picture in deciding the question of compensation in simple pneumoconiosis. It might be fairer to workers in dusty occupations if we were to compensate respiratory disability regardless of the radiological picture whenever this disability falls well outside the range of the normal at the appropriate age and is not clearly attributable to other non-occupational respiratory disease. Improvements in respiratory function tests, which might enable the pattern of impaired function in pneumoconiosis to be distinguished from that of emphysema and chronic bronchitis, would greatly help in this decision.

Reference

1. Davson, J. and Susman, W. (1937). J. Path. Bact., 45, 597
2. Harding, H.E. (1949) Proc. 9th Int. Congr. Industr. Med. 1948
London 692.

February 27, 1953

Carl M. Peterson, M.D.
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Seward E. Miller, M.D.
Medical Director
Chief, Division of Occupational Health
Federal Security Agency
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Dear Doctors:

Before leaving today on a lecture tour through South America, I wish to acknowledge and thank you for your letter of February 24 with regard to the problem of definition of pneumoconiosis, discussed during the Monday morning session of The Seventh Saranac Symposium. You will hear more from me regarding this on my return to the Laboratory the latter part of April.

With every best wish,

Sincerely yours,

Arthur J. Vorwald, M.D.
Director, The Trudeau Foundation
and The Saranac Laboratory

AJV:gn

DEFINITION AND CLARIFICATION OF TERMS PERTAINING TO PNEUMOCONIOSIS

I shall discuss the questions put to me by Dr. Vorwald in ^{his} letter.

The Significance of the word Pneumoconiosis

Strictly, pneumoconiosis means a "process in the lungs caused by dust" and is theoretically initiated when the first dust particles become lodged in the lung parenchyma and cause any reaction. For practical purposes the definition of pneumoconiosis proposed by the International Conference of the I.L.O. in Sydney in 1950 is reasonable: "Pneumoconiosis is a diagnosable disease of the lungs produced by the inhalation of dust, the term 'dust' being understood to refer to particulate matter in the solid phase but excluding living organisms". The term 'diagnosable' here implies the presence of signs or symptoms, but not always loss of function (paras. 5 and 27 of the report of the Conference). To me, the word 'disease' here implies any abnormal condition of the lungs. It is always difficult to define just where normality ends and abnormality begins. No sharp distinction can be drawn between the accumulation of soot or dust in a town dweller's lung and the earliest changes of coal pneumoconiosis, or even silicosis (1,2). It would be reasonable in some cases to speak of 'town dweller's pneumoconiosis'. In scientific medicine the diagnosis of pneumoconiosis should thus be made as soon as any characteristic change can be detected, whether this be clinical or radiological during life, or histological after death, so long as this change can be reasonably attributed to the presence of dust. In most forms of pneumoconiosis histological changes are the first to appear, and radiological abnormality precedes the appearance of clinical or functional changes by many years (although in some forms of pneumoconiosis such as asbestosis and byssinosis the reverse is true). These radiological changes are due to dust in the lung and must for scientific and logical reasons be regarded as constituting pneumoconiosis.

I feel there is some danger in using a definition based upon histological examination of lungs because it may encourage men to undergo lung biopsy in order to obtain histological proof. Without lung biopsy a histological definition cannot be logically applied during life.

The only objection to applying the term pneumoconiosis to cases without disability is psychological. A man may be told that he has pneumoconiosis on the grounds of radiological evidence, and may then be disgruntled if he be not granted compensation: or, in an area such as South Wales where large numbers of men are seriously affected by the disease, he may be alarmed by the diagnosis and develop an anxiety state. From this point of view it would be advantageous to have a separate term which would be used for non-disabling pneumoconiosis, but since non-disabling and disabling pneumoconiosis are usually but different stages of a single pathological process, it would be illogical in scientific medicine to use separate terms. Moreover, radiologists or pathologists, as they classify radiographs or sections of lungs, cannot distinguish the disabled from the non-disabled and must apply the term pneumoconiosis to every case in which they find abnormality attributable to dust.

In the Pneumoconiosis Research Unit we have not found it difficult to explain to a man that his radiograph "shows that he has been a miner, but does not show enough dust to do any harm" and that he cannot therefore expect compensation. In border-line cases also we have found no difficulty in explaining that doctors may disagree with one another in assessing the severity of the disease so that in such cases, while application for compensation may be worth making, the man must not be surprised if it fails despite our own diagnosis of pneumoconiosis. (We have found that the Welsh coal miner is much more ready to accept the existence of observer error among doctors than are members of the medical profession themselves!) For this reason I do not think that there is any need to introduce a new term for non-compensatable pneumoconiosis.

For purposes of compensation it may be necessary to define a limit of acceptable abnormality (whether clinical or radiological) below which an official diagnosis of pneumoconiosis will not be made, and which may lie above the lowest limit recognised by special investigators. It is for legislators to decide what they want to have compensated - disability, loss of earning capacity, shortening of life expectancy or what they will. It is then up to doctors to attempt to devise means of measuring the factor that is to be compensated, and to show the degree of accuracy with which it can be measured. At present legislators often demand, and doctors are prepared to give, expressions of opinion which cannot be supported by scientific evidence, particularly in relation to respiratory disability.

We have also to consider whether a man can be disabled by pneumoconiosis without developing characteristic radiological signs. In South Wales Professor Gough has occasionally demonstrated coal miners' lungs with severe focal emphysema which must have been the cause of pulmonary disability during life but which were unassociated with the radiological abnormality characteristic of pneumoconiosis. Such cases, however, are very uncommon. Pulmonary disability attributable to dust inhalation may also arise in byssinosis and asbestosis without any characteristic radiological changes. Dr. Hugh Jones will show in his paper tomorrow that, in the sample of miners and non-miners that we have studied, the miners without any radiological signs of pneumoconiosis appeared on the average to have impaired ventilatory capacity when compared with non-miners of the same age. This impairment was in some cases associated with a tendency to bronchial spasm. Whether this disability is attributable to dust inhalation we cannot yet say. The sample was a small one and the results may not be generally applicable. Further studies of much larger samples of miners and men working in other dusty and non-dusty occupations will be required to establish whether coaldust inhalation can disable without causing radiological changes.

Our preliminary findings do suggest that we may at present rely too much upon the radiological changes in deciding compensation for disability in simple pneumoconiosis. Until we know more about the relationship between disability and severity of radiological change from studies on large populations of miners and ex-miners (not on hospital populations, men applying for compensation or working populations, which are biased in relation to disability) we shall not be able to say what emphasis should be placed on disability measured by clinical or physiological methods and what emphasis should be placed on the radiological picture.

Active and Inactive Dusts

The pathological activity of a dust depends not only on its chemical nature but also on its physical characteristics, such as size distribution, and upon the dose administered, so that no simple classification of dusts into active and inactive is possible. For example, a given quantity of 20 Angstrom silica may be highly active if given in a single large dose, but the same quantity may have no effect at all if administered in small concentrations over a longer period.

It might be possible to reach a theoretically sound classification of dusts according to their activity on the basis of the relationship between the amount of material retained in the lung and the degree of pathological change.

Thus, an active dust would be one in which the degree of pathological reaction to its presence in the lung is great in relation to the amount of retained material (e.g. silica and beryllium) and an inactive dust would be one in which the pathological change is relatively small (e.g. coal and iron), but here we meet the difficulty that dusts such as coal which would be classified as inactive per se may, in combination with tuberculous infection, produce gross pathological changes.

Any classification of dusts into active or inactive on the basis of animal experiments may be dangerously misleading. Coal dust was for years regarded as harmless in Great Britain and is still so regarded in Germany, largely because animal lungs do not show any fibrotic response to inhaled coal dust, and yet the inhalation of coal dust has disabled more than 20,000 coal workers in South Wales during the past twenty years.

Silicosis

Dr. Vorwald asked me whether silicosis is a "disease" or a "condition". I don't see any difference in the meaning of these two words. I regard any pathological change attributable to free silica as a form of silicosis. I distinguish the formation of uniformly distributed concentric collagenous fibrotic nodules by the term "Classical nodular silicosis".

Terms for Specific Entities

The word silicosis should only be used when it has been clearly demonstrated that the pathological changes in the lung are attributable to the action of silica. So far as I know this can only be done where the dust exposure is to pure silica or where the reaction in the lung is of the classical type. For all other pulmonary disease due to dust the word pneumoconiosis should be used, preceded by the name of the dust or occupation by or in which the disease is caused. The term "anthraco-silicosis" should be applied only to those cases of classical silicosis in which there are also deposits of coal in the lungs (for example, rock workers in coal mines) but should not be applied to the ordinary coal workers' pneumoconiosis in which there is no evidence that silica is responsible for the fibrosis.

Clarity is gained in referring to the pulmonary conditions attributable to specific dusts by using prefixes to the word pneumoconiosis, e.g. soot pneumoconiosis, talc pneumoconiosis etc.

1. Davson J. and Susman, W. (1937), J. Path. Bact., 45, 597.
2. Harding, H.E. (1949) Proc. 9th Int. Congr. Industr. Med. 1948 Lond. p.692.

Discussion

DR. VORWALD: It would appear that Dr. Fletcher would like to restrict the word pneumoconiosis to those conditions and dusts which produce disease. He does not accept the assumption that there are inactive dusts but considers all dusts to be active depending, of course, upon concentration. He believes that if the concentration of any dust is high enough, certain clinical signs and perhaps symptoms will appear in individuals exposed to the dust. Have I stated your views correctly, Dr. Fletcher?

Fletcher's corrections
DR. FLETCHER: ~~Yes~~ No. Some dusts are more fibrogenic than others, but all insoluble dusts may cause pneumoconiosis if inhaled in sufficient quantity.

DR. VORWALD: Dr. Fletcher deprecates the use of the term anthraco-silicosis and therefore is in agreement with some other panel members. He would use the word silicosis but would restrict it to those conditions where the presence of silica, crystalline free-silica, in the lung is proven. But how do we prove that silica is present in the lung? Radiologically? By biopsy and chemical analysis of the tissue? By the presence of silica in the sputum or in the urine? Are we to rely only on the occupational history of exposure to silica and, ipso facto, conclude that exposure necessitates the presence of silicosis? Are we to discard the observation that there is individual variation in the way men respond to inhaled free silica, that some men can be exposed for long periods of time and never develop silicosis?

*Revised
GW*

Remarks by A. J. Orenstein, M.D.

*...sterilized ...
...with ...
...in ...*

I am very glad.....
.....
.....or processes concerned.

Discussion

DR. LANZA: I don't believe there is as much disagreement between us as we might have expected. I differ with Dr. Sander on the broad use of the word pneumoconiosis and I believe considerably more thought must be given to the question of active dusts and inert dusts. We always think of kaolin as an inert dust yet Dr. Kenneth Lynch recently presented me with the facts on two fatal cases involving exposure to kaolin. He had the lungs for study. Of course, it was a question of dosage. These two men had ^{each} worked 47 years in the industry and had been almost smothered in kaolin. To quibble as to whether that kaolin dust is an active dust or an inactive dust does not get us very far.

DR. GREENBURG: I would like to emphasize two points. The first point is the change in our attitude toward many types of dust formerly considered to be completely inert. Years ago we believed silica to be the only villain, and I remember when the pathological effects of inhaled talc dust were attributed to the few per cent of free silica in the dust. Today we know that to be untrue and therefore I say we must keep an open mind and not assert that any particular dust is completely free of damaging effect.

The second point, one which I emphasized before and which Dr. Orenstein mentioned, is the fallaciousness of reading too much into an X-ray picture. You may look at an X-ray picture of the chest and see no nodulation, then examine an X-ray picture of the lungs after they have been removed at autopsy and blown up and you will frequently discover that nodulation is present. The X-ray picture is sometimes most deceiving. The character and extent of the pathology in the lungs, the respiratory incapacity and the early roentgenographic manifestations of dust inhalation bear no close relationship to

each other. I admit we may have no substitute for the X-ray but we must be very careful in our interpretation based on the roentgenogram alone and must try to use also careful industrial histories, studies of the dust, and studies of the functional capacity to work.

DR. SANDER: I should like first to correct one mis-statement. It is theoretically possible, of course, to inhale enough inert material to drown one's self, but we have not seen in industry, at least in our area, concentrations of dust sufficient to have that occur. We have seen extreme deposits of tin and of iron in welders. Those welders had a terrific exposure inside small tanks, yet none of them had symptoms, neither pulmonary symptoms nor symptoms elsewhere. None had a measurable decrease in the maximal breathing capacity; all had perfectly normal lung function. Therefore in industry, at least out our way and so far as I know in this country, we have not had sufficiently high concentrations of inert material to produce disability or to cause any impairment of lung function. It is theoretically possible, of course, for very high concentrations to be responsible for impairment of lung function and Dr. Fletcher cited one case in which there was probably an excessive concentration.

For the overall use of pneumoconiosis I still believe in conforming to the generic meaning of words. I don't like to call an entity something it isn't, yet we propose to limit pneumoconiosis to a fibrotic pneumoconiosis. If that limitation be accepted, then another term will be needed to identify the dust conditions which are truly benign and non-fibrogenic.

MR. WAGNER: From the legal standpoint the important thing is, what diseases do we wish to make compensable? For what diseases should industry bear a responsibility for compensation? We should fit the terms closest

to those diseases so as not to leave to the courts the interpretation of those terms.

DR. FLETCHER: I think Dr. Sander has misunderstood me; I do not wish to restrict the term pneumoconiosis to fibrotic changes. Pneumoconiosis is a process in the lung and, theoretically, that process is initiated when the first dust particle is engulfed by the first lung cell. But I hesitate about a statement that a dust ^{cannot injure the lung, based on} ~~is inert on the basis of~~ ^{cases without} two or three ^{patho-}logical ^{study.} ~~cases.~~ The statement may be true, but I am a little disturbed about it because of ^{our} experiences ^{with coal dust, which was always considered harmless to the} ~~related to coal.~~ lung until extensive studies of coal miners and coal trimmers were carried out. I shall have to disagree with Dr. Lanza that a pneumoconiosis must be occupational. What would we say about a deep sea diver whose hobby was carving sandstone and who got pneumoconiosis from his hobby?

DR. HUSSEY: I would like to emphasize that we are discussing a subject about which our knowledge is most imperfect. Definitions at their best are imperfect and in trying to formulate a definition of pneumoconiosis we have a very complicated situation.

Since many people refer to Zenker as the originator of the word pneumoconiosis, I would like to point out that Dr. Zenker was a pathologist and was not necessarily introducing a term. In almost the last paragraph of his paper entitled The Pulmonary Diseases Due to Inhalation of Dust he stated that rather than use that long-winded expression he would simply use the word pneumoconiosis, which has the same meaning as the title. Also, he was writing about an anatomical disease while all of you are talking about clinical disease.

The derivation of many words in medical terminology is more remote than that of the term pneumoconiosis; for example, typhoid fever is a term we derive from comparative and statistical data and tuberculosis is a name derived from the appearance of tubercles. The term pneumoconiosis must

mean, if we accept the way it was introduced, disease of the lung due to the inhalation of dust. And since this is one of the few instances in medicine in which our definitions have to stand a legal test - always a difficult matter - it is important to have in our definitions not just vague words but very definite expressions to indicate as nearly as possible the exact meaning we wish to convey - whether an anatomical meaning or a clinical meaning or a combination of the two. Finally, the definition should include some statement that permits one to determine, insofar as is possible, the disability, particularly its extent, associated with the pneumoconiosis.

DR. VORWALD: Dr. Fletcher pointed out that as soon as a particle of crystalline free silica is deposited in the lung there is a cellular reaction, *either one cell or a host of cells depending upon the local concentration of the dust.* However, the individual may present no evident clinical signs or symptoms and the chest radiograph may appear normal. Quartz may be recovered from the lung, thus indicating that the individual inhaled free silica. Pathologically, the lung may exhibit various degrees of cellular reaction, but without the nodular lesions of silicosis. The point here is that the lung manifests tissue reaction to inhaled silica. Obviously, a diagnosis of silicosis cannot be made, since the definition of silicosis requires the presence in the lung of nodular shadows that can be visualized radiographically or of nodular lesions in the lung. Thus, by definition the individual does not have silicosis, but what does he have? *↑ correct*

DR. BRODKIN: In an individual whose roentgenogram shows nodulation but who has no symptoms, is there or is there not loss of function? As a thoracic surgeon I sometimes take out half a lung yet the patient, with all that tissue removed, is symptomless from the standpoint of pulmonary disease. I should like to cite one case, that of a man of 21 who had been

BY DOCTOR FLETCHER:

Mr. Chairman and Gentlemen: I'm in slight difficulties, since this is the first occasion on which I've attempted to take part in a panel of this kind and I don't know to what extent I'm expected to answer the questions that Doctor Vorwald fired at me across the Atlantic, or to what extent I can throw brickbats at the-- some of the remarks which I regard as highly debatable, which have been made already this morning. I will attempt to combine the two.

I have been pleased with the definition of

pneumoconiosis proposed by the International Labor Office Conference in Sidney two years ago, that pneumoconiosis is a diagnosable disease of the lungs produced by the inhalation of dust, the term dust being understood to refer to particulate matter in the solid phase but excluding living organisms.

The term diagnosable is later amplified in the report as meaning the presence of signs or symptoms, with radiological abnormality as being a sign, but not always loss of function. To me, the word disease implies any abnormal condition of the lungs.

Now, it's difficult to define where normality leaves off and where abnormality begins. You can't draw any sharp distinction between the accumulations of soot dust in a town dweller long in a smoky city such as Sheffield, and the early stages of pneumoconiosis, and, of course Zenker and Schuller describe pneumoconioses in the town dwellers in the apex.

But, I should think here, we want to increase the acceptance within the framework of medical knowledge of the statistician and if we do that, possibly even the lawyers may follow us and recognize statistical concepts, and I regard as abnormal, something which is two standard deviations away from the normal, and where we can measure that, we should admit that as an abnormality or come to some

agreement as to what the number of standard deviations we can admit are.

We can do that by surveying the normal population and discovering the frequency of certain radiological pictures, comparing them with the occupation we're interested in, and we may even say the pathologists to start, can become quantitative in their work so that they can follow suit with the radiologists and the physiologists. The physicians -- well, perhaps later, they too.

With regard to the pathological definition, that, of course, is useless in life, and I do deplore, I must say, for legal purposes, a definition based on fibrosis which can not be diagnosed without lung biopsy, because much as I like my colleagues, the lung surgeons, I think they already have a tendency to be too enthusiastic.

For purposes of compensation, for scientific purposes, I think we must be quite strict and recognize a process in the lungs attributable to dust. Now, that is pneumoconiosis, whatever the lawyers want us to say. It is up to the lawyers to decide what they want to compensate. Do they want to compensate a man for having an abnormal radiograph or for being disabled? Let them say what it is they want to compensate. Then the doctor, in his term, can say how he will set so sort of limit, with, I plead, the help of the statistician.

One of the difficulties of the conflict between the compensatable pneumoconiosis and scientific pneumoconiosis is psychological. The man who has pneumoconiosis, diagnosed on a radiograph by one doctor, applies to compensation and is turned down and is then disgruntled, or in an area like South Wales, where there are hundreds - yes, tens of thousands - of disabled men with pneumoconiosis, he develops an anxiety state.

Well, I suggest that clever doctors needn't be baffled by this any more than they are by tuberculosis. If we diagnose, on a radiograph, a hemolytical scar, we don't tell the man he has got tuberculosis. We ignore it, but we make a record in our own notes that he has had tuberculosis; he has some sign of tubercular infection. The same sign can be used in pneumoconiosis, and we have not found it difficult to explain to miners that they have dust in their lungs, but that is not compensatable pneumoconiosis.

Most miners have signs of dust in their skin where they have wounded the skin or have got dust in the skin. We can explain it is not a dust disease; it is dust in the skin. They have dust in the lung and they will accept that explanation.

Moreover, we have found the miner much more willing to accept the fact that two doctors may disagree in their opinion about an X-ray film than our doctors in service.

Active and inactive dusts, I don't think you can make any distinction without so much specification that the distinction loses any value. In the case of silica, a clearly active dust, if given in high dose, the effect will be clearly shown, but the same quantity spread over a long period of time will have no effect whatever on an animal's life. Active or inactive? It depends on the dose.

And, for that reason, I am very much afraid of Doctor Sander's statement about inactive dusts, that soot is inactive. Krause and Gartner, recently in Germany, have confirmed the observations of Locke, that men working exposed to high concentrations of pure soot, 99.9 percent soot, pure carbon, developed radiological abnormalities, and I have seen them myself, identical with coal gas. Is soot radiologically inactive? No, not if it's inhaled in so much quantity that it displaces air from the lungs; solid material up against air in the lungs will cause a radiological picture, so soot is as radiologically opaque as edema fluid when it replaces that in the lung, and you see the shadow, and I don't like the quotation of two or three cases of tin oxide, barium oxide lungs and iron in the lungs as evidence that no matter how much of these dusts these men inhale, they would suffer no damage.

I could match Doctor Sander's pathological pictures with coal miners lungs, with just about as much

dust, just about as much reaction. All right, would argue Doctor Sander, there is no such thing as a dangerous quantity of coal dust. Coal is an inactive dust. Let him come to South Wales and see what happens when the man inhales enormous amounts or quantities of coal over long periods of time. Then, indeed, there is a reaction.

And, I suspect that these diseases due to the so-called inert dusts have their character determined very largely by the quantity of dust that is inhaled, and that any inert dust or so-called inert dust, simply because it is not silica, as the usual meaning for the term, any such inert dust inhaled in sufficient quantities, will cause liability to disability, especially when complicated by tuberculous infection.

Doctor Vorwald asked me, whether silicosis was a disease or a condition. I'm afraid that these two words have very little difference in their meaning in Great Britain. It's one of these meanings that are different, of course, because we think we talk English and you think we talk English and actually I think our languages are, in some respects, different. But I would simply value the distinction, if it is necessary, of classical nodular silicosis as one particular form of silicosis, with the particular process in the lungs strictly attributable to the inhalation of silica, by which I mean crystalline free

silica, and that is silicosis, and I think that silicosis as a term, should only be used, or the word should only be used as a term when silica can be quite positively, by irrefutable scientific evidence, shown to be the cause of the condition, and that is, as Doctor Sander said, why I object to the term anthraco-silicosis. It pre-supposes that silica has something to do with the pathology and leads to dangerous concepts, such as in Germany, where they are expressing all the stone dusts in their mines, because it contains silica. Thereby, they hope to prevent the anthraco-silicosis which is being caused by the coal dust in their mines, to which they pay still, no attention. A very dangerous term, anthraco-silicosis.

One short point, I want to make, on the active and inactive dusts, and that is I do deprecate very strongly, any attempt to determine the activity of a dust on animal experiments or rather the inactivity of a dust. Coal dust, in our experience, has practically no pathological reaction in the lungs of animals at all, but it has disabled well over 20,000 coal miners in South Wales in the last twenty years.

One last point, and that is pneumoconiosis without radiological change. In coal pneumoconiosis, Professor Goff has shown lungs obviously severely injured by coal dust, in which, during life, there has been no radiological

abnormality, and Doctor Hugh-Jones will show you, in his paper, tomorrow afternoon, I hope, that coal pneumoconiosis is very simply related, until massive fibrosis intervenes. In simple pneumoconiosis with just a few mottlings, there is no relation between the severity of the radiological change and the disability.

In Great Britain, a man has to show radiological abnormality during life to be admitted to compensation, and I think that it might perhaps be fairer to admit any miner who shows a significant deviation from normal pulmonary function to compensation, regardless of the radiological change. In that way, a few men who would be disabled by ordinary emphysema and bronchitis, had they not been coal miners, they would be given compensation, but personally, I would prefer that, ethically, from - to the present system which certainly debar some men who are disabled by coal dust from being admitted to compensation.

Thank you.

(Applause).

BY DOCTOR VORWALD:

It seems to me we're really rolling along now. We're getting to the point where the noon hour is coming, but yet we're approaching the most interesting part of this discussion. I attempted to say to you, let's go without our lunch and carry on, but I don't think that would be

agreeable to all of you.

It would appear, from my interpretation of what Doctor Fletcher said, he should like to restrict the word 'pneumoconioses' to those conditions, to those dusts which produce disease, but I gather that he does not recognize that there are inactive dusts, that all dusts are active, of course, depending upon, as he said, concentration. If the concentration is large enough, then there will appear certain clinical signs and perhaps symptoms in this individual who has been exposed to those high levels of concentration of any dust. Am I correct, Doctor Fletcher?

BY DOCTOR FLETCHER:

Yes.

BY DOCTOR VORWALD:

* He also would like to emphasize that he deplores the use of the term 'anthraco-silicosis' and thus is in agreement with some other members of the panel. He would use the word 'silicosis', but restrict it in a sense to those conditions where the presence of silica, free crystalline silica is actually proven clinically.

Then, I ask the question, again, for consideration, how do we prove that silica is present in the lung? By biopsy? Can we see it radiologically? Is it in the sputum? Do we find it in the urine? Or must we rely upon the occupational history, that this man has had an exposure?

A. J. Weinstein

Symposium
Paper

TENTATIVE

INTRODUCTION TO DISCUSSION ON
DEFINITIONS AND CLARIFICATION OF TERMS CONCERNING
PNEUMOCONIOSIS.

SEVENTH BARANAG LAKE CONFERENCE.

22nd September, 1952.

I am very glad the conveners of this Conference included definitions in the subjects for discussion. In dealing with industrial diseases, and particularly the pneumoconioses, unambiguous, clear and precise definitions are of special and very practical importance. On the definitions may depend the grave issue of compensation, and thus the economic future of the claimant and, on the other hand, the prosperity or failure of an industry.

Definition of an industrial disease presents problems beyond those of defining a disease from the clinical-pathological point of view.

There is necessarily an inter-relation between the two problems, and it lies principally in the concept of what is "disease".

Thus, to take as an example the definition adopted by the Sydney Silicosis Conference, 1950, which is also the one recently adopted by the South African Bureau of Standards:-

"Whilst in a wide sense the term 'pneumoconiosis' connotes any condition of the lungs resulting from the inhalation of dust, that might or might not, be of clinical significance, for the purpose of the International Labour Organisation the Conference accepted the following definition:-

-Pneumoconiosis -

See
this comment
in letter 846
Aug 27/1952

Pneumoconiosis is a diagnosable disease of the lungs produced by the inhalation of dust, the term 'dust' being understood to refer to particulate matter in the solid phase, but excluding living organisms."

Immediately the question is posed: "What does 'disease' really mean?" This word may be defined, and has been defined, variously, depending very much on the context in which it is used. The pathologist would say it means deviation from normal of the cells of the tissue. But about a century ago Virchow pointed out that cellular abnormalities do not necessarily connote functional abnormality. In fact, normal function may be continued in an organ the cells of which show abnormality, and function may be impaired in an organ in which cellular abnormality is not detectable. There are gradations, of course, in the inter-relations between observable cellular states and function.

The clinician might define disease as impairment of bodily functions.

In considering industrial diseases the problem of compensation must be borne in mind. The concept of disease is, therefore, usually related to functional impairment. The terms commonly employed are "incapacity" or "disability". I think in industrial disease legislation there is only one exception to this usage, and that is in the South African Silicosis Act, in which compensation is provided for a condition in which no functional impairment need be demonstrated, or even be claimed by the applicant.

Having regard to the course of the majority of pneumoconioses, it is hardly surprising that the definitions so far adopted have all, so far as I can ascertain, caused difficulties to those concerned with the diagnosis, certification and assessment of compensation, the latter being generally based on the degree of disability/incapacity certified by the examiners.

South African experience, where medical examinations and assessment of degrees of silicosis have been centralised in one establishment for over 35 years, provides probably more promising information than any other experience, for reviewing the effect of definitions on the problems of assessing the existence and stages of the disease, and the consequent compensation.

The first Act, that of 1912, had a very simple definition. This Act was entitled "The Miners' Phthisis Act, 1912". The definitions were:-

"Miners' Phthisis shall mean silicosis of the lungs", and

"... the benefits to be awarded under this Act shall be -

(a) to a miner who shows definite physical signs of miners' phthisis and whose capacity for underground work is thereby not seriously or permanently impaired, the sum of eight pounds per month for a period not exceeding one year;

(b) to a miner who has contracted miners' phthisis in a marked degree and whose physical capacity for underground work is thereby seriously and permanently impaired, the sum of eight pounds per month, but not exceeding four hundred pounds in all: Provided that where a monthly payment has been awarded to a miner under this sub-section the Board may, if it is satisfied that the special circumstances of the case warrant it, extend the period of such payment so that the payments made to such miner will exceed four hundred pounds. All such cases shall be specially reported on by the Board under section twelve of this Act."

-There-

There was no mention of tuberculosis in this Act.

The next phase in making definitions was in the 1916 Act. In this Act the definitions were:-

"(2) For the purposes of this Act the expression 'miners' phthisis' means silicosis of the lungs; and tuberculosis means tuberculosis of the lungs or of the respiratory organs. A person shall, for the purposes of this Act, be deemed to be suffering or to have suffered from miners' phthisis in the primary stage if, though he shows or has shown definite physical signs of that disease, his capacity for work is not, or has not been seriously and permanently impaired.

A person shall for the purpose of this Act be deemed to be suffering or to have suffered from miners' phthisis in a secondary stage, if the miners' phthisis shows or has shown itself in a marked degree and thereby his physical capacity for work is or has been seriously and permanently impaired."

Now tuberculosis is included and is compensable, whether this disease is or is not associated with silicosis. The importance of this amendment of the previous Act lies in the onus thrown on the examiners to decide whether the pulmonary tuberculosis is or is not associated with silicosis, inasmuch as the compensation payable was very much higher when silicosis was also present - nearly 2½ times higher.

It will be noted that only two stages of silicosis were defined.

In the 1919 Consolidating Act radical changes were made in the definitions. The definitions now were:-

"(2) For the purposes of this Act the expression 'silicosis' means silicosis of the lungs. A person shall, for the purposes of this Act, be deemed to have or to have had silicosis -

(a) in the ante-primary stage when it is found by the Bureau that physical signs of damage to the lungs short of definite physical signs of silicosis have become evident and that such damage has supervened during and in consequence of employment in a scheduled mine;

(b) in the primary stage when it is found by the Bureau that definite and specific physical signs of silicosis are or have been present and that capacity for work is or has been impaired by that disease though not seriously and permanently;

(c) in the secondary stage when it is found by the Bureau that definite and specific physical signs of silicosis are or have been present and that capacity for work is or has been seriously and permanently impaired by that disease."

Attention is invited to the following important

changes:-

(a) The introduction of the ante-primary stage. (Later it was renamed 1st stage).

(b) The use of the expression "definite and specific signs of silicosis" in the definitions of the "primary" and "secondary" stages, but "short of definite physical signs of silicosis" in the ante-primary stage. The word "specific" is here omitted.

The examiners were now required to make up their minds as to what are "definite physical signs of silicosis" and what is meant by "specific signs", which the law did not require to be present in

ante-primary stage. To a medically trained audience there is no need to labour the point that the almost impossible was asked. Later, I will allude to the unsatisfactory - to use the mildest term - consequences of these ill-thought out definitions.

At this stage I think it well to say a word about why the ante-primary stage was introduced. The reasons given were:-

- (a) That by eliminating from hazard men in the earliest detectable stage of silicosis, progression would be arrested and the victims with still practically undiminished functions would rehabilitate themselves in some other employment;
- (b) That the liability in respect of compensation would in the long run be much diminished.

Of course, in the event neither of these expectations was realised to any notable extent.

My views on the 1919 definition of ante-primary silicosis were stated before a parliamentary committee in 1920:-

" I should like the Committee to pause and consider exactly what these sentences mean. If they mean anything at all they mean this: The Law directs the medical examiners of the Bureau to say that a man has silicosis before there are any facts capable of being established by any method of diagnosis with which we are acquainted to-day or which are likely or may even reasonably be expected to be discovered in the future, to indicate that there is any silicosis whatever. It is difficult and to me impossible to understand how such a law can be laid down, except on the supposition that people are still labouring under the medieval belief that medical men possess occult and supernatural powers which are beyond the ken of ordinary men."

-Then-

Then came the 1925 Act.

Here the definitions

read:-

"A person shall for the purposes of this Act be deemed to have or to have had silicosis -

(a) in the ante-primary stage, when it is found by the Bureau that the earliest detectable specific physical signs of silicosis are or have been present; whether or not capacity for work is or has been impaired by such silicosis;

(b) in the primary stage, when it is found by the Bureau that definite and specific physical signs of silicosis are or have been present, and that capacity for work is or has been impaired by that disease, though not seriously and permanently;

(c) in the secondary stage, when it is found by the Bureau that definite and specific physical signs of silicosis are or have been present, and that capacity for work is or has been seriously and permanently impaired by that disease or when it is found by the Bureau that tuberculosis with silicosis is or has been present."

Particularly to be noted are the words "... the earliest detectable specific physical signs..." in the ante-primary stage, and also the addition of the words "... whether or not capacity for work is or has been impaired by such silicosis". The word "specific" is now added to the definition of this stage.

Here an attempt was made to clarify the ambiguity of the previous definitions. But, has it been done? In the light of subsequent experience, I do not think so. At a recent inquiry into the working of the Silicosis Medical Bureau, the Chairman of the Bureau categorically stated that the certification of the first stage of silicosis is a "fifty-fifty matter". The import of this statement need not be underlined to an audience such as this.

-The-

The next and last changes in the South African legal definitions are in the Silicosis Act, 1946:-

* 'silicosis' means any form of pneumoconiosis due to the inhalation of mineral dust; and for the purposes of this Act a person shall be deemed to be or to have been suffering -

(a) from silicosis in the first stage (which corresponds with the ante-primary stage under the 1925 Act) when the Bureau has found that the earliest specific signs of silicosis, detected by any means whatsoever, are or were present in the lungs of the person in question, irrespective of whether his capacity for work has or has not been impaired by the said disease; or

(b) from silicosis in the second stage (which corresponds with the primary stage under the 1925 Act) when the Bureau has found that moderately marked specific signs of silicosis are or were present in the lungs of the person in question and that the said disease has not incapacitated him from performing moderate manual work; or

(c) from silicosis in the third stage (which corresponds with the secondary stage under the 1925 Act) when the Bureau has found that marked specific signs of silicosis are or were present in the lungs of the person in question and that the said disease has incapacitated him from performing moderate manual work."

Here we have some important changes:-

(a) Silicosis means any form of pneumoconiosis.

This means that for legal purposes - compensation, etc. - any pneumoconiosis is to be dealt with as if it were silicosis, and the definitions of the three stages of silicosis apply to the other pneumoconioses. Previously only silicosis -

strictly - was compensable.

(b) The definition of the first stage (the old ante-primary) is

-elaborated -

elaborated in the words "detectable by any means whatsoever", in place of the previous "earliest detectable", and the word "physical" is omitted.

(c) "Capacity for work is or has been impaired by that disease" is replaced by "... the said disease has not incapacitated him from performing moderate manual labour" in the definition of the second stage.

(d) In the third stage "capacity for work is or has been seriously and permanently impaired by that disease" is replaced by "... the said disease has incapacitated him from performing moderate manual labour". The last sentence of the 1925 definition relating to the presence of tuberculosis is deleted. Tuberculosis is dealt with separately in the 1946 Act as follows:-

" 'tuberculosis' means tuberculosis of the respiratory organs and a person shall, for the purposes of this Act, be deemed to be suffering from tuberculosis -

(a) if the Bureau has found the said person's sputum to contain tubercle bacilli; or

(b) if the Bureau has found the said person to be suffering from 'closed' tuberculosis which seriously impaired his working capacity."

One of the effects of these definitions on certification is that the words "specific signs" are taken to mean radiological signs-

signs only, and assessment of stages has been made virtually solely on the radiological appearances. The contention appears to be that the radiograph provides the only "specific signs" of silicosis: That in the absence of certain radiographic appearances, any cardio-pulmonary disability present must be due to some other cause; and that it is not the function of the examiners to seek for the "other cause".

One consequence is that the miners assert that they "lost confidence in the Silicosis Medical Bureau" and have threatened to boycott it if the personnel and methods are not radically changed forthwith.

Because of the attitude of the miners, the Government recently appointed a Commission of Enquiry. This Commission in its report submitted to Government the following definitions of silicosis. ("Silicosis" means any pneumoconiosis in this context).

"A person shall be deemed to be or to have been suffering -

(a) from silicosis in the first stage when the Bureau has found during life the earliest signs of silicosis demonstrable by radiological or any other appropriate examination, or when the Bureau has found demonstrable permanent impairment of respiratory or cardio-respiratory function; or when the Bureau has found post-mortem early lesions of silicosis or chronic bronchitis and/or its sequelae. Provided that the impairment of the respiratory or cardio-respiratory function or the chronic bronchitis and its sequelae shall, in the opinion of the Bureau, be permanent and have resulted from the performance of work in a dusty occupation and shall have been found within a period of twelve months since the person last worked in a dusty occupation.

(b) from silicosis in the second stage when the Bureau has found by radiological and other appropriate examination that silicosis has given rise to impairment of respiratory or cardio-respiratory function causing reduction in capacity for exertion to the extent of unfitting the person for more than moderate exertion, or the Bureau has found post-mortem such lesions of the cardio-respiratory organs due to silicosis as would have incapacitated the deceased for more than moderate exertion during life;

(c) from silicosis in the third stage when the Bureau has found by radiological and other appropriate examination that silicosis has given rise to impairment of respiratory or cardio-respiratory function causing reduction in capacity for exertion to the extent of unfitting the person for more than light exertion, or the Bureau has found post-mortem such lesions of the cardio-respiratory organs due to silicosis as would have incapacitated the deceased for more than light exertion during life."

The Commission remarked:-

"This definition has been drafted with the view to removing as much ambiguity as is possible and at the same time leaving the Bureau the same amount of latitude in exercising the science and art of diagnosis as must be left to those called upon to deal with a subject which is by no means either static or easy.

The words 'specific', 'moderately marked' and 'marked' have been deliberately omitted from the proposed definition as being vague and too much the subject of a variety of personal interpretation, and because their use tends to over-emphasize the radiological findings and to obscure the overwhelming importance of incapacity for work.

Furthermore, the words 'cardio-respiratory' are brought into the definition in order to indicate as clearly as may be that such affections of the lungs as are usually present in at least the later stages of silicosis cannot be dissociated from cardiac disfunction.

The word 'exertion' has been substituted for the words 'manual work' in order to indicate that what is to be assessed is loss of power for sustained muscular effort."

-Turning -

turning to definitions adopted or proposed elsewhere than in South Africa -

The 1930 International Conference, convened by the International Labour Office, in Johannesburg, adopted the following definitions:-

"Silicosis is a pathological condition of the lungs due to inhalation of silicon dioxide. It can be produced experimentally in animals."

As to stages of the disease -

"In the 'first stage' symptoms referable to the respiratory system may be either slight or even absent. Capacity for work may be slightly impaired. There may be a departure from the normal in percussion and in auscultatory signs, and the radiograph must show an increased density of linear shadows, and the presence of discrete shadows, indicative of nodulation.

In the 'second stage', there is an increase of the physical signs observable in the 'first stage', and the radiograph shows an increase in the number and size of the discrete shadows indicative of nodulation with a tendency to their confluence. There must be some degree of definite impairment of working capacity.

In the 'third stage' all the above conditions are grossly accentuated and indications of areas of massive fibrosis are usual. There is serious or total incapacitation.

Pulmonary tuberculosis may be present in any of the above described 'stages' of silicosis, altering the symptoms, physical signs and radiographic appearances, and the degree of working capacity. Its presence must therefore influence the 'stage' classification of the individual, which classification must in these circumstances be based more on the degree of loss of working capacity than on physical signs and radiographic appearances."

The concept that "silicosis" is due to silicon dioxide, accepted by this Conference, has, of course, been modified. It is now accepted that comparable lesions and damage can be produced by certain silicates. However, purists may contest this.

In 1934 the Committee on pneumoconiosis of the Industrial Hygiene Section, American Public Health Association, defined silicosis as:-

"a disease due to breathing air containing silica (SiO_2), characterized anatomically by generalized fibrotic changes and the development of silicary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present), and by characteristic X-ray findings."

Sayers states that pneumoconiosis produced by other dusts were purposely omitted (U.S. Public Health Reports, 49, 1934).

I have already quoted the definition adopted at the Sydney Conference.

This Conference also considered a scheme for the classification of radiographs of pneumoconiosis.

The scheme is largely the work of Dr. Fletcher and he is so much more competent to deal with this subject, that I merely mention it, in the hope that he will present it for discussion. But I should like to suggest that in discussing this matter we should be quite clear that radiological findings do not provide a criterion for assessing disability. The impression

-that-

that the radiograph is the criterion has bedevilled the subject.

There remains only to remind you of the terminology attached to the various pneumoconioses. The Sydney Conference deprecated the extension of terms, and suggested that the terminology should in future take the form of naming the dust to which the worker is exposed in the industry or process concerned.

27th August, 1952.
AJO/EAC.

Then are we to discard the observation which I think is valid that there is individual variation in the way men respond to free crystalline silica, that some can be exposed for longer periods of time and never manifest evidence of silicosis?

I put the question again: If, then, silicosis is to be used and to be restricted only in instances where free crystalline silica - the presence of free crystalline silica can be proven, then how do we prove it?

The discussion of these papers, before we again turn the discussion back to the panel members, and to the audience, will be led by Doctor Orenstein, who perhaps comes from the greatest - from the greatest distance away, to join us in this Symposium. Doctor Orenstein is Chief Medical Officer of the Rand Mines in Johannesburg of the Union of South Africa. Doctor Orenstein.

BY DOCTOR ORENSTEIN:

Mr. Chairman, Ladies and Gentlemen: I'm afraid that any attempt on my part to lead this discussion would be to take up much more time than you have at your disposal, and strain your patience beyond almost endurance.

If you followed at all what has been said up to now, you will have grasped the extreme complexity of this question. I have had very little time to prepare for this,

BY DOCTOR ORENSTEIN:

Mr. Chairman, Ladies and Gentlemen: I'm afraid that any attempt on my part to lead this discussion would be to take up much more time than you have at your disposal, and strain your patience beyond almost endurance.

If you followed at all what has been said up to now, you will have grasped the extreme complexity of this question. I have had very little time to prepare for this,

because my masters gave me very little notice about coming here and I didn't really know at the time, what was expected of me. However, I thought that perhaps I might take up a side of the question which is the - from my point of view, a practical one, and that is definition from the point of view of the person who has to certify for compensation, that is to say the question which was in some respect dealt with then by Mr. Wagner.

If one is to attempt to deal with the point, with the definition itself, from the pathological and clinical point of view, strictly medical point of view, one enters a very wide field indeed. To attempt to add to the definitions, the importance of exact definition from the certifying physician's point of view, is indeed a very wide field also.

Now, South Africa is a little, small country, but it has had experience with legislation on pneumoconiosis or strictly speaking, silicosis, of some forty years today, and there it was the experience in legislation, of attempts at definition of terms, over numerous acts, as Mr. Wagner mentioned in the days of certain of the states, where again and again, attempts were made to clarify the position for the benefit of the certifying physician.

The first question that arises immediately is, of course, what does one mean by disease. That, in itself,

is a problem which could be discussed at considerable length. From the clinical - pathological point of view, of course, it was laid down a century ago, by Wilhelm, that it is an abnormality of cells, but you can have abnormality of cells, as you all know, in an area, or in an organ without having any disability whatsoever, and, therefore, disease, in the sense of the person who has to decide that something exists in an individual for which he has some - for which he has to receive compensation.

We, I think, or those of us who have to consider legislation from the point of view of compensation, must bear in mind two important and somewhat opposing points of view. One is that if you don't define a disease, an industrial disease, with great exactitude, or with as much exactitude as you can manage, grave injustice would be done or might be done on the one hand, to the man who suffers from the disease, such as has been the case on a large scale in South Wales, or you might, in the case of certain industries, damage the prosperity of that industry by excessive compensation being paid out, and therefore, in our concept, in South Africa, we consider pneumoconiosis, silicosis, an incapacity of some sort, a disability of some sort.

Now, the legislation of South Africa has started with the Act of 1912, when it was first thought that disease existed which was due to quartz, and at that time, two

stages of disease were defined and at that time, it was, of course, called 'Miners' Phthisis', and the first definition was: "Miners' Phthisis shall mean silicosis of the lungs". Certain benefits are awarded when there is no serious or permanent impairment, or more if there is serious or permanent impairment. No mention was made of tuberculosis.

Four years afterwards, it was - the legislation was amended, and it was then said that, for the purposes of this Act, the expression 'Miners' Phthisis' means silicosis of the lungs, and tuberculosis means tuberculosis of the lungs or of the respiratory organs.

Now, here it may be strange to an American audience to appreciate how the acts are drafted. In our draft, you start off by defining in the beginning of the act, every term that you use, as if it were a dictionary, and it is that definition that fixes the attitude of the Compensation Board toward the particular individual's troubles.

Now, in 1916, tuberculosis was included and became compensable, whether associated with or without silicosis. Then, it was realized that the definition which was used then was not satisfactory, and a much more elaborate one was introduced three years afterwards under the so-called Consolidating Act, and a radical change was made.

At this stage - in this Act, a third stage of silicosis was recognized and that is the so-called ante-

primary stage, now called the first stage.

The ante-primary stage was then defined, when it is found by the Bureau, that is to say the medical examiners, that physical signs of damage to the lungs, short of definite physical signs of silicosis - SHORT OF DEFINITE PHYSICAL SIGNS OF SILICOSIS - have become evident, and that such damage has supervened during and in consequence of employment in a scheduled mine; and then the primary stage, with a certain amount of disability, and the secondary stage with more disability. I have no time to read these without detaining you too long.

Now, the ante-primary stage was introduced when a condition, ill-defined condition became compensable. The reason for that I don't want to go into at the moment beyond saying in a word that the idea was then that if you remove the man from exposure to dust early enough, the disease would not progress and, therefore, the man would be able to locate himself in some other occupation, and incidentally, the people who were responsible for paying the compensation would be relieved of the higher compensation which, in the secondary stages, meant pension for life and a pension to the widow or dependents. That, of course, in the event neither of these expectations proved to be correct. We all know now that at least in relation to SiO_2 , no matter how early you remove a person, a certain progression

takes place.

In 1920, in giving evidence before a Parliamentary Commission that dealt with this subject, I expressed very strongly the view that they barked up the wrong tree and that they passed legislation which, in itself, was not reasonable and that they expected medical men to possess occult or supernatural powers in making a diagnosis of something which didn't exist.

Then, in 1925, an act was passed in which the ante-primary stage was defined, the earliest detectable specific signs - physical signs, of silicosis, and thereafter, the whole of the compensation question became bedevilled by this word 'specific', because the interpretation was made that specific meant radiological, radiological and nothing else.

Well, this went on for some considerable time until it became clear, as was said a while ago by several speakers, that the radiological signs do not necessarily or, for that matters, frequently even, bear any relation to disability, and so it came about last year, that the Miners Union memorialized the Government and said that unless a radical change is made in the function of the Government Bureau of Medical Examiners, they would boycott the Bureau. The effect of that would have been a general strike, of course, because it is a crime to employ any person in a

dusty occupation in South Africa who is not possessed of a certificate of being free from silicosis and tuberculosis, which he has to supply at periodic intervals.

Now, as a result of that, a Commission was appointed under the chairmanship of a Judge of the Supreme Court, and they made certain recommendations with regard to alterations in the act. Meanwhile, something had to be done to placate the Miners' Union, and rapidly, an amendment to the Silicosis Act was passed at the last session of Parliament early this year, or the middle of this year, in which amendment - an amendment was passed named 'Pulmonary Disability'.

Pulmonary Disability was defined very loosely as something or other which a man might acquire by working in a dusty occupation, but which is not silicosis, but which disables his lungs or his heart or lung and heart. That was passed while the Commission was in session and before the Commission was ready to advise with regard to the best way of amending the act and the definition.

Well, that particular amendment will probably be thrown out at the next session of Parliament and a new definition will be introduced.

I - you already had quoted to you the Sydney definition of pneumoconiosis with which I entirely agree. I thought I told you briefly about our difficulties, because they are difficulties which apply or might apply to anybody

in any country that attempts legislation of dust disease of the lungs, damage to the lungs due to working in dust.

We have - the Commission of which I happen to be a member, felt that the definitions should be to the effect that -- I'll read to you just this much if you'll forgive me; it won't take more than a few minutes -- it first of all stated in the definition of terms that the word 'Silicosis' as used in the Act means any pneumoconiosis whatsoever, the word silicosis, so you turn this around and talk of a pneumoconiosis, but then Mr. Wagner and some of the lawyers here realize that when you have had a number of previous acts and you have to make an amendment, it is always dangerous to change the name of the act, because the effect - it might effect previous decisions of the court on previous compensation, so the particular technique of saying silicosis means pneumoconiosis, you might just as well have said if the word 'John Smith' is used in this Act, it will mean pneumoconiosis, it will have the same effect.

And they defined it in these stages or proposed to the Government that the three stages be defined as follows:

That a person shall be deemed to be or to have been suffering -- that is to say on post-mortem -- from silicosis:

(a) In the first stage when the Bureau has found during life the earliest signs of silicosis demonstrable by

radiological or any other appropriate examination, or when the Bureau has found demonstrable permanent impairment of respiratory or cardio-respiratory function; or when the Bureau has found post-mortem early lesions of silicosis or chronic bronchitis and/or its sequelae. Provided that the impairment of the respiratory or cardio-respiratory function or the chronic bronchitis and its sequelae shall, in the opinion of the Bureau, be permanent and have resulted from the performance of work in a dusty occupation and shall have been found within a period of twelve months since the person last worked in a dusty occupation.

Then, it proceeds to define the second and third stages on the basis of incapacity for exertion.

The Commission remarked, in defense of its proposal, that "This definition has been drafted with the view to removing as much ambiguity as is possible and at the same time leaving the Bureau the same amount of latitude in exercising the science and art of diagnosis as must be left to those called upon to deal with a subject which is by no means either static or easy.

"The words 'specific', 'moderately marked' and 'marked' have been deliberately omitted from the proposed definitions as being vague and too much the subject of a variety of personal interpretation, and because their use tends to over-emphasize the radiological findings and to

obscure the overwhelming importance of incapacity work--
for work.

"Furthermore, the words 'cardio-respiratory' are brought into the definition in order to indicate as clearly as may be that such affections of the lungs as are usually present in at least the later stages of silicosis cannot be dissociated from cardiac disfunction.

"The word 'exertion' has been substituted for the words 'manual work' in order to indicate that what is to be assessed is loss of power for sustained muscular effort."

Now, finally I just want to say one word in emphasis of what has been said before, and because of our rather unfortunate experience, and that is we must bear in mind the danger of trying to use the radiograph as a measure of disability, as a measure of disability, as a measure of a man's impairment of function, except in a very highest degree of fibrosis; it is no measure of disability whatsoever and should not be used as such.

I have seen in many law suits, as long as I have experienced, and it is a long experience, any number of people, and such people brought before the Commission, who radiologically, showed practically no signs, very very early signs of striation or slight nodulation, who were invalids in every sense of the word, hardly able to do anything, certainly would have been handicapped if you asked

them to walk rapidly down this room, and we have seen people with advanced nodulation, massive fibrosis of portions of the lung, who are playing a very good game of tennis, and those are extremes, of course, but what I suggest to you gentlemen is that, after all, we, as industrial medical men, must be deeply concerned with the question of doing substantial justice to the people who look to us, as medical men, to advise those who draft legislation which may mean the prosperity of a person or, at least may mean a person not being thrown into the utmost depths of poverty, because he - because somebody quibbled about the wording of a law or somebody has quibbled about what should be done to establish that man's ability to earn a living, or on the other hand, his entitlement to compensation for something that has happened whilst he was directly or indirectly in the service of the nation.

We can not escape that, and to satisfy ourselves merely with purely medical, clinical, pathological definitions, does not - is not substantial justice to the task, the duty which is thrown upon us as industrial medical men.

(Applause).

BY DOCTOR VORWALD:

The meeting is now ready for the rebuttal, may I say, by the members of the panel who have heard one another

express their views, and the first one to call upon is Doctor Lanza. Do you have anything to add, Doctor Lanza, any other remarks?

BY DOCTOR LANZA:

I don't think I have anything to add; I don't think there is as much disagreement between us as we might have expected. I differ with Doctor Sander on the broad use of the word 'pneumoconiosis' and I think a lot more thought has to be given to the matter of inert dusts or active dusts. I don't know whether Doctor Kenneth Lynch is in the audience or not, but not long ago, he presented to me the facts on two cases, two fatal cases of kaolin. Well, now, we always think of kaolin as an inert dust, yet here were two men who have died. He had the lungs, so he knew what he was talking about.

Well, of course, it comes back, just as one of the speakers said, to the question of dosage. These two men had worked forty-seven years in the industry, each one of them, and they had little - they had been smothered or drowned in kaolin. Now, to quibble as to whether that is dust, inert or active dust, I don't think, gets us very far. I don't think I have anything else to add.

BY DOCTOR VORWALD:

Doctor Greenburg?

BY DOCTOR GREENBURG:

Mr. Chairman, I don't think there is any great area of difference between the speakers here this morning. I think perhaps some of us have one or two points of disagreement with Doctor Sander, but I think that will all be straightened out after we get the record and read it over and discuss it a little more between ourselves.

I would only like to emphasize two points. I first came to Saranac in about 1921, if I remember correctly or possibly '22. Doctor Gardner was then just beginning to get interested in the dust question and I had been working for a year or two on the Public Health Service. I started in April, 1918 to be exact, and I came up here and we spent time in the laboratory doing some foolish things. Among others, we put salicylic acid down the nostrils of some guinea pigs to see what happened. Of course, you know what happened, most of them drowned, but anyway, one thing that did emerge over the period of years was this, that I've seen a change in our attitude toward many different types of dusts which, at the beginning, we thought were completely inert.

At that time, silica was only supposed to be the only villain in the picture, and I remember the time when the question of talc dust, the effect of talc dust was brought up and it was said that the pathological effects of respiration of talc dust were due to three - two or three or four percent of free crystalline silica, and today we know that

to be completely untrue, so I would say that anything we do, we must keep an open mind and not say that this or that dust is completely free of damaging effect, because time has proven us wrong again and again and again, the last time with the coal dust studies in South Wales.

And, the other point which I should like to emphasize is this point which has been mentioned by Doctor Orenstein, in which I believe I mentioned before, and that is the fallaciousness of attributing, reading too much into an X-ray picture. If you look at an X-ray picture of the chest and you see that there is no nodulation present, and then wait until the autopsy of the lungs, taken out and blown up, and take an X-ray picture of the lungs, you will very frequently see nodulation present there, and I say that the difference between the early stages of dust inhalation and incapacity, bear no co-relation to each other, just as Doctor Orenstein has said, and that the X-ray picture is sometimes most deceiving, and I admit we may not have any tool to substitute for it, but we must be very careful in our interpretations based on X-ray pictures and try instead to use the X-ray picture, careful industrial histories and laboratory studies on the dust and studies of the functional capacity of the work. Thank you.

BY DOCTOR VORWALD:

Doctor Sander?

BY DOCTOR SANDER:

I seem to be in the minority on the overall use of pneumoconiosis, and before I talk about that, I would like to correct one misstatement. I fully agree that you can inhale enough, I mean it's theoretically possible to inhale enough inert material to drown yourself; there is no question about that. We have not seen, in industry, sufficient concentrations of dust to have that occur, at least not in our area, and we have these extreme deposits of tin and iron in the welders, for example. Those welders have terrific exposure, inside of small tanks, and yet none of them had symptoms, none of them have had symptoms elsewhere. None of them had a measurable decrease in the maximal breathing capacity, perfectly normal lung function, so that in industry, out our way at least, and as far as I know in this country, we have not had sufficiently high concentrations of inert material to cause any impairment of lung function or disability. I say it's theoretically possible, of course, and Doctor Fletcher cited one case where it probably was such an excessive concentration.

But, for the overall use of pneumoconiosis, I still believe in staying with the generic meaning of words. I'm a purist in that regard. I don't like to call something what it isn't, and here we are, going to limit a pneumoconiosis to fibrotic pneumoconioses, and what do we call these

others which we have been able to show, and many others have shown have been truly inert. I throw out that question and I think maybe we might get an answer before the week is over.

BY DOCTOR VORWALD:

Mr. Wagner? Dick?

BY MR. WAGNER:

Well, I'll just say one sentence from here. I don't think there is anything else I can add to what I've said. I think, from the legal standpoint, the important thing is, as has been pointed out, what diseases are we going to make - what do we want to make - compensable? What are the diseases for which industry should bear a responsibility for compensation? And then let's see if we can fit the terms closest to them, so as not to leave the courts to construe those terms.

BY DOCTOR VORWALD:

Charles?

BY DOCTOR FLETCHER:

I think Doctor Sander has misunderstood me. I don't want to restrict the term 'pneumoconiosis' to fibrotic changes. Pneumoconiosis is a process in the lung, and I think, theoretically, that process is initiated when the first dust particle is engulfed by the first lung cell. You can't get away from it, that's when the process is first

initiated. It becomes done, of course, when Doctor Vorwald can say there is a deviation from normal; I can say there is a significant deviation; Doctor Hugh-Jones can say there is a deviation, clinically, but you can say they are all pneumoconioses, right from the engulfment of the first particle by the cell. I want to make that quite clear.

All I hesitate about is the statement that a dust is inert on two or three pathological cases. It may be quite true, I'm not denying it is true, I'm just quite frightened of it because of similar experiences in relation to coal.

The other point that I'd like to make, and that I tried to make earlier, is that I do strongly disagree with Doctor Lanza that a pneumoconiosis must be occupational. Perhaps he has a vested interest there. What would we say to a deep sea diver whose hobby was sandstone and who got pneumoconiosis from his hobby?

BY DOCTOR VORWALD:

There is an announcement that Doctor Parker is wanted on the phone, call 154, Doctor Parker.

That brings us then to participation from the floor. Now, what are your questions, what are your comments? Doctor Hussey?

BY DOCTOR HUSSEY:

I'd like to say a word or two about one or two of

the matters that I made notes about. I don't want to be - I don't want to appear to be - I'm not, but I don't want to appear to be pedantic about this.

In the first place, I would like to emphasize that it has been - or give further emphasis to the fact that we are discussing a subject about which our knowledge is most imperfect. Now, does anybody disagree with that? Definitions at their best are imperfect, so we have a very complicated situation to try to formulate a definition.

Then I'd like to say a word in defense of Doctor Zenker. So many people used the word 'pneumoconiosis' and refer to Zenker as having originated it. I'd like to call your attention, if you don't know it, to the fact that Doctor Zenker was a pathologist and he was not necessarily introducing a term. If you will read his paper, you will find the very last sentence, almost in the last paragraph, the title of his paper was "The Pulmonary Diseases Due to Inhalation of Dust", and he said rather than to use this long-winded expression, I will simply use the word 'pneumoconiosis', which means the same thing. That's the way the Greeks say it, and I won't go into the Greek about it; you can find some Greek scholar for that, but that's the dope.

Now, he was talking about an anatomical disease, not clinical disease. All of you are talking about clinical disease. Don't, then, blame it on Zenker. If he were here

today and he knew the story about Mary's little lamb that went to Pittsburg, I'm sure he'd speak of it that way.

Now, also in making these various definitions, we have to keep in mind that, in most instances, if you look into the origin of our terms that we use in medicine, they are much less secure than is the term pneumoconiosis, which I think must be, if you're going to accept the original way in which it was introduced, mean diseases of the lung due to the inhalation of dust.

Typhoid fever is a term we derive from comparative and statistical data. Tuberculosis is a disease that we derive from the appearance of tubercles, and finally, I'd like to indicate to you, the need, in thinking about all of these matters of definition especially, and this is one of the few, if not the only instance in medicine, I don't mean the pulmonary dust diseases, but occupational diseases, where our definitions have to stand a legal test and that's always a difficult matter, but I do think it's very important for us to keep in mind that we must have in our definitions, not just vague words, but very definite expressions to indicate as nearly as is possible, the exact meaning that we wish to convey, whether it's an anatomical meaning or whether it's a clinical meaning or whether it's a combination of the two, and then finally, since the one consideration that's of particular importance involved in all of these matters,

is the question of disability, the definition should also include some statement that permits one to determine, insofar as it's possible, the disability and the extent of it in connection with that disease.

BY DOCTOR VORWALD:

Doctor Fletcher cited the fact, in agreement with Doctor Sander, and I saw Doctor Sander shake his head, that as soon as a particle of free crystalline silica is deposited in the lung, there is a cellular reaction. In studying these many cases which come to us here, there is always the problem, in my own mind, and that is looking at tissue, I see that cellular reaction. Maybe one particle of free crystalline silica, maybe more particles. There is a reaction there of the lung to free crystalline silica. However, there has never been any evident clinical signs or symptoms. The radiograph appears normal; there are no respiratory symptoms. We know the man has been exposed to free crystalline silica. We can recover free crystalline silica from the lung which may or may not mean a thing, and the real problem then is, does this man have silicosis. I am aware, as you are, of our definition of silicosis, that if a man is in the presence of brief nodular shadows, radiographically, and nodular lesions in the lung; yet here we have this case where frequently silica particles are in cells within the lung and there is cellular damage, yet

what we see does not conform to our definition. Does this man have silicosis? Those are some of the problems.

Again, it is open to the floor. Doctor Brodman?

BY DOCTOR BRODMAN:

May I speak from here? I am one of these intrepid thoracic surgeons that Doctor Fletcher referred to in a very hesitating manner. I can not help but think, we speak of looking at an X-ray, with nodulation, and since there are no symptoms, is there loss of function or isn't there? Now, sometimes it becomes my duty to take out a half a lung. Now, there is no question at all in that case, that there is loss of pulmonary tissue, or I might say destruction of pulmonary tissue and still that patient without, or with a half a lung gone, is absolutely simulous from the standpoint of pulmonary disease.

I should like to cite one case to which Doctor Fletcher referred, and that is a case of a young man of 21 who had an exposure ti silica, and had diffused nodulations throughout both lungs, and six experts gave six different diagnoses. I must confess it remained, it was left to me as the thoracic surgeon to take out a small piece of lung which did not involve too much surgery or excessive danger to the patient, aside from the fact that he was quite dismayed, and somewhat anxious, but with that piece of lung, we were able to very, very definitely determine that the man

had sarco-silicosis.

BY DOCTOR VORWALD:

Doctor Merewether, would you care to say anything? I call upon Doctor Merewether because, during recent days, we have had considerable discussion about these things, and Doctor Merewether whom you will hear later on in the Symposium is also a guest at the Symposium coming from England.

BY DOCTOR MEREWETHER:

Mr. Chairman, Ladies and Gentlemen: The most expert exposition of this subject this morning has, as I expected, served not to show grievance so much, as to throw up differences in the various aspects that the people have to deal with in this matter. I have not prepared anything on it, and I feel rather like the new tenth husband of the famous film star, in a very humble and embarrassed position, but more particularly, because - and more particularly because I feel that I can't give you any answers.

Doctor Raymond Hussey implies that the Greeks may have an answer to these things, but I think we should better get down to the real priorities in this discussion, as much as possible, as must be obvious to all of us. The important thing is or the important things are really - first priorities are prevention and compensation for those unfortunate enough to get the disease, and its nomenclature and definitions which enable these matters to be facilitated

forwardly and these are more important than academic protection, and moreover, we can't expect total prevention of these diseases, but only a proportion, and you, no doubt, will come along way in this in the course of the future, but we must try and keep up to these things in practical priorities.

Therefore, since - since the word 'pneumoconiosis' in the industries and amongst the people who suffer from the disease, has a most sinister meaning, I deplore the use of the word except in medical circles, to indicate something that does not cause disablement, sickness or death. Now, it's unfortunate really, that the word has come to stay. It's been in existence so long, but it is very necessary for operational purposes that we should know what it means, as all the speakers have pointed out.

Now, there are three types in general, of people who consider or who have to consider the word 'pneumoconiosis'. The widest conception of it is that of the man who is not the least bit interested, the intelligent layman, the doctor who is specializing in some other - highly specialized in some other line of business, who uses it merely meaning a dusty lung, but it sounds better to call it 'pneumoconiosis'.

The second is a wide definition and a particular definition like that adopted - adopted by the ILO, some years

age. This is important that the ILO has to adopt a high definition, so as to enable, in your somewhat infrequent discussions that they have dealing with particular subjects like pneumoconiosis, there is opportunity to expand, follow expert knowledge and focus research and compensation laws and prevention laws, and so forth, in the right direction.

The third and more narrow types of definition are those which we commonly use in different forms in different states in different countries. They will probably always vary for other reasons, amongst themselves. In England, we have a rather tight definition, which in itself, has varied since 1925, 1925, the first main definition of these things, and that is likely to vary again as more knowledge comes along.

At the present moment, pneumoconiosis is defined as fibrosis of the lungs due to silica dust, asbestos dust or other dust, and includes the condition known as dust reticulation of the lungs.

Now, that is for a particular purpose. It is something that expert chest physicians in your country, my country, and so forth, understand what it means, because they have seen so many cases and they can apply it with justice. The word 'other dust', if there was another dust, then it would have to be scheduled separately and that is what is causing the trouble at the moment. Berylliosis at

at the moment, for instance, is presently being compensated as beryllium poisoning.

Now, it is important to have a definition for compensation purposes, which can be applied by specialists in the field, for the reason that compensation is something for a - in this case, it's something for a devastating disease, and those people should be paid equivalent for it, those that get it. It is just to compensate them for such a disease.

It is also unjust to compensate somebody who has not got the disease, by making the definition too wide. In our law, we also have a method, a provision in the Industrial Injuries Act which provides for prescribing a disease with retrograde effect, a very unusual thing in any legislation, that enables one, as soon as evidence is found of the occupational character of any disease, evidence that satisfies the criteria just mentioned, it can be scheduled and people who have got the disease, after it is, you know, on the list, can be compensated.

Now, I said I was going to add a few bits and pieces to the difficulties. Is carcinoma due to actual trioxide dust, or nickel, if nickel is the cause, dust, or the bichromates pneumoconiosis. This is not a pneumoconiosis by our own definition, so we have to put it in the Acts separately.

Now, mixed dust, some people suggest we should put the dust on, but here surely we should have further information from the experts in minerology, and geology, because you could have mixed dusts which are deliberately mixed, and they produce, as everybody knows, very mixed types of X-ray appearances. Then you can have mixed dusts which are mixed in some areas of the country and not in others. Thus, you can have a pure talc, or so-called talc, French chalk, call it what you like, which has no free silica in it.

Yet, I have seen, I remember the last time I was here ten years ago, through the courtesy of my old friend, that great man, that lovable great man, LeRoy Gardner, a slide of a lung which in the literature was labeled talc pneumoconiosis, and it was pretty obvious that the man died of silicosis, and asbestosis together, whatever else the talc did. It was obvious to anybody. He didn't label it that.

Now, the quiet agreement that a thing is called inert, may be illustrated by Roy Gardner's magnificently illuminating deliberations, that dust if inhaled, may cause damage to the lung in spite of being classed as active or inert dust, and lastly, the main dust that causes disability in disease, we know, and if we can get a nomenclature suitable for them, we have achieved a priority, which will cause

or help justice to be done to those known cases. It may do a little damage to the researchers who are looking into the epoch type, but it is more important to see that justice is done and do it quickly.

BY DOCTOR VORWALD:

Are there other questions or comments from the floor? (No response).

We have had a discussion of disease, what is disease. Anyone wish to comment as to their interpretation of a disease. Doctor McCann, really, you should say something about it. Doctor ^{Waring} Werner, care to make a remark?

BY DOCTOR McCANN:

This is a very difficult question to which to address oneself. As a clinician, I think we have to relate the word disease to the occurrence of symptoms and we must realize, at the outset, that disease in the sense that symptoms occur, is not so extensive that a - that it becomes a pathological process.

A very skilled athlete may develop a system of distress if the emotional distress is great enough, so I think that we have then to take into consideration the factor of great stress, the factor of normality or abnormality of the individual; we have to take the individual as a whole into account, in regard to his ability to adapt himself to stress. He may have a pathological process in one organ,

for which he will comensate by the adapted processes in another, so that under the same stress, he may react differently from his neighbor. I think it's one word to try to define disease as distinct from a pathological process; he would have to take these things into account, aside from an individual with definite pathological process recognized as being produced by silica, who may never be put under stress sufficient to bring out symptoms, so that from a clinician's standpoint, I think he would have to say then that disease is the difference in adaptation to stress produced by a pathological process, to such an extent that the individual is unable to adapt with ease to the extent that a normal man would do so. This is very difficult.

BY DOCTOR VORWALD:

We will get some remarks here. We're going to close the session immediately because we are late. But there is considerable agreement between our thoughts concerning definitions. However, I think that all of us will agree that there is considerable disagreement also, and that there are terms which we are - which we use today which need definition and clarification, and thinking about this, anticipating that we would come out of this discussion with something that might not be concrete enough, I thought that it might be well to make something concrete out of

this discussion, and in debating this thing with my colleagues, it was thought that we better present this to the Symposium, that this Symposium regrets the confusion caused by the use of the word 'pneumoconiosis' with uncertain meaning and that this Symposium invites Doctor Seward Miller, Doctor Peterson of the Industrial Counsel, Doctor Miller of the United States Public Health Service, to constitute a small committee, to constitute a small committee under the heading of representatives from bodies and individual experts specifically concerned with the use of the term 'pneumoconiosis', to consider its definition, having regard to its application in the research, clinical, operative, and legal fields, with the aim of common interpretation, and that that committee should report.

What is your comment, do you think that such a committee should be constituted under the aegis of a group having interest, such as the industrial counsel, or on industrial health, or the United States Public Health Service, to appoint this committee and this committee will consider these terms and that there shall be letters - there shall be also corresponding members to the committee, our friends from England, our friends from South Africa and elsewhere, who might write their views to the Committee. Is that worthwhile? I think it is. Any expression?

BY DOCTOR PAUL RICHARDS:

I make a suggestion that your suggestion be made as a motion and I second the motion.

BY DOCTOR VORWALD:

My suggestion - it has been suggested that my suggestion be made a motion and that that motion be seconded. It has been seconded. So it has been done, so is it your wish then, by a voice merely, that this be done, and then I think Doctor Seward Hiller and Doctor Peterson in the audience, and I shall place the burden of responsibility upon either one or both of them to arrange such committee. Is that your wish by voice vote? Those in favor? (Response of ayes). Those against it? (No response). I take it it's unanimous, then, Doctor Peterson.

BY DOCTOR PETERSON:

I'd like to see in that representation, the American Public Health Association.

BY DOCTOR VORWALD:

American Public Health; I'm sure Doctor Seward Miller is here and Doctor Peterson and they have heard your recommendation, and they will take consideration of it.

BY DOCTOR GREENBURG:

Mr. Chairman, the American Public Health Association now has a committee at work on it.

BY DOCTOR VORWALD:

Well, perhaps the American Public Health Associa-

ANTHONY J. LANZA, M.D.
Chairman
New York City

WARREN F. DRAPER, M.D.
Washington, D. C.

LLOYD E. HAMLIN, M.D.
Chicago, Ill.

UTHERFORD T. JOHNSTONE, M.D.
Los Angeles, Calif.

ROBERT A. KEHOE, M.D.
Cincinnati, Ohio

HENRY H. KESSLER, M.D.
Newark, N. J.

COUNCIL ON INDUSTRIAL HEALTH
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Port Huron, Mich.

WILLIAM P. SHEPARD, M.D.
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JAMES S. SIMMONS, M.D.
Boston, Mass.

JAMES H. STERNER, M.D.
Rochester, N. Y.

535 N. DEARBORN STREET, CHICAGO 10, ILLINOIS

February 24, 1953

Dr. Arthur J. Vorwald
Director, The Saranac Laboratory
and The Trudeau Foundation
P.O. Box 551
Saranac Lake, New York

Dear Doctor Vorwald:

During the Seventh Saranac Symposium, at the session on Definitions and Clarification of Terms Pertaining to Pneumoconiosis, held on Monday morning, September 22, 1952, your records will show that the problem of definition of pneumoconiosis was referred for study to Seward Miller and C. M. Peterson.

This matter has been given due consideration. We are most favorably impressed with a statement prepared by Leroy U. Gardner, as follows:

"I prefer to retain the original meaning and use pneumoconiosis as a generic term to describe all forms of pulmonary reactions to inhaled dust, with no implication as to character, severity or effect on function." (The Pathology and Roentgenographic Manifestations of Pneumoconiosis. Leroy U. Gardner, M.D., JAMA 114:535-545, Feb. 17, 1940.)

According to our present view, we have seen no good reason for attempting to improve on this concept.

Sincerely yours,

Seward E. Miller
C. M. Peterson

PART TWO

The Inhalation of Certain Industrial Substances

Chapter Two ~~Two~~ 2

VORWALD COLL
BOX 89-91

Introduction

Theodore F. Hatch

As I listened to the program dealing with definitions and terminology and to the various views expressed, it occurred to me that some of the differences made evident by the discussion stem from the fact that we as a group do not have as complete an understanding as we would like to have of the mechanism of action of ~~the~~ various dusts. As we gain in our knowledge of the basic mechanisms of action the problem of defining the particular condition we wish to discuss may become easier.

The series of papers which follow pertains to the inhalation of certain industrial substances. The first papers review briefly various aspects of some of the materials which have received considerable attention in the past; about those materials there has accumulated a great deal of information which, though far from being complete, furnishes a broad background of facts and experience. Another paper will be devoted to new industrial products - to the recently-developed synthetic silicas which have become quite important in commercial applications. Finally, some of the newer concepts regarding the fate of dust particles that have been inhaled into the lung will be discussed. The way in which particulates are handled in the course of inhalation, their receipt by the lungs, the amount of retention - an understanding of all these things is fundamental to a better comprehension of the whole problem of dust diseases.

We sometimes wonder whether the day will ever come when we shall be able to anticipate the difficulties associated with the inhalation of particulate matter and shall not have to wait until the difficulty has developed before becoming concerned about it. Our success in meeting this problem will depend upon the degree to which we can reduce our understanding to fundamentals, to the common denominators that go to make up the problem, whether it be one of asbestos or of bauxite or of something else.

Is it going to be possible for us to anticipate and to predict, from basic studies of new dusts, the possibility of toxic reaction? It would be a discouraging situation if one believed that we could never achieve that objective. We may not be able to explain everything but I certainly hope that in the future we shall be able to do a better job in anticipating possible trouble than we have in the past. And in obtaining a better understanding of these problems we must depend as much on epidemiological findings from the field as we do on the findings from the laboratory. The one complements and supplements the other.

tion should be a tri-part members of this group, with Doctor Hiller and Doctor Peterson to formulate a definition under the aegis of individuals, specifically concerned with the use of terms. Then I place that responsibility upon Doctor Seward Hiller, Doctor Peterson, hoping they can arrange this committee.

(Adjournment of morning session 12:20 P.M.)

SUBJECT: THE INHALATION OF CERTAIN INDUSTRIAL SUBSTANCES

Chairman: Theodore F. Hatch

Monday, Sept. 22.
2:00 - 5:30 P. M.

Brief Reviews

Silica

Thomas M. Durkan

Asbestos

Arthur J. Vorwald, M. D.

Beryllium

Harriet L. Hardy, M. D.

Bauxite

C. G. Shaver, M.D.
and

Donald Solandt, M. D.

Discussion.

New Synethetic Silica

Philip C. Pratt, M. D.

The Fate of Inhaled Particulates

Merril Eisenbud

Discussion, led by

Theodore F. Hatch.

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Discussion, led by

Theodore F. Hatch

BY DOCTOR VORWALD:

We're behind schedule, so I think we should begin. We have a very interesting afternoon. We don't really wish to run overtime as we did this morning. I hope all of you have found food. The Chief of Police called me up and told me that there were a number of cars parked about without having paid their nickels, but yet they do not have the red tag on their windshields. Again, we urge that you register at the John Black Room and receive your automobile cards, otherwise, you may find yourself elsewhere other than in the Symposium.

Our topics for this afternoon concern various substances and we have selected a Chairman whom we believe is outstanding as a moderator of those topics, Doctor Hatch from the School of Public Health, the University of Pittsburgh and the Industrial Hygiene Foundation of the Mellon Institute in Pittsburgh. He will serve as the Chairman. Doctor Hatch.

BY DOCTOR HATCH:

Doctor Vorwald and the Symposium: It occurred to me as I was listening to the program this morning, the discussion of the various views, that some of the differences that were made evident by the discussion stem from the fact that we, as a group, do not have as complete an understanding as we would like to have of the mechanism of - of the various dusts, and that as we gain in our understanding of

the basic mechanisms of action, the problem of defining the thing that we're talking about may become easier, and it would seem to me that our afternoon session, therefore, should contribute in a very useful way, to easing that difficulty as it appeared in the morning discussion.

We have, as you all see, a series of talks on The Inhalation of Certain Industrial Substances, beginning with the old one of silica and going through to some of the newer materials and some of the newer concepts with regard to the action of dust in the lung.

I understand that some of the people in the back of the room this morning had difficulty in hearing the various speakers, so that I will now ask the speakers this afternoon to keep their voices up, keep that in mind, and to make sure that they speak up loudly.

I was provided by Doctor Vorwald with a program in advance, in which he has carefully noted the number of minutes allowed for each speaker. Since we are a little late in starting, I shall pound the bell down here very promptly to make sure that they stay within their time.

The first subject this afternoon, under this general heading of inhalation of certain industrial substances, is a discussion by Mr. Durkan of the Laboratory Staff on Silica. I'm sure Mr. Durkan needs no further introduction than that to us. Mr. Durkan.

BY MR. DURKAN:

Mr. Chairman, Members of the Symposium:

(Doctor Durkan read his prepared paper, which is on file at the Laboratory).

BY DOCTOR HATCH:

In view of the fact that our several subjects in this topic are expressed in several papers, it would be best to perhaps limit discussion and comments to the end of the presentation, so I call next on Doctor Vorwald who will discuss the findings in reference to asbestos.

BY DOCTOR VORWALD:

It's not easy to cover the topic of asbestos and asbestosis in the short time which we must, and I shall only try, and shall try to be very brief.

As you perhaps know, the disease asbestosis was first described about 1900 in England and since that time, there have been many, many reports concerning asbestos and notably those reports by Doctor Merewether in England, Doctor Lanza and McConnell and Fennel of the United - of the Metropolitan Life Insurance Company; by Doctor Lynch and others, and again by Doctor Lanza in the Monograph, published by the Oxford University Medical Publications, entitled "Silicosis and Asbestosis".

And, following these reports, it was thought that

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And, following these reports, it was thought that

we knew all about asbestosis, that there was nothing more that could be known. However, I don't believe that that view is substantiated and, certainly, all of us recognize that there are still many, many problems concerning the disease asbestosis, problems which are very complex and problems which must still be considered.

Therefore, in my remarks, I certainly do not wish to appear dogmatic in any way. I will merely try to point out some of the things which are established and seem to be established, and other things which appear to be valid in accordance with our present knowledge.

You all know what asbestos is. Of course, the name is not really one that refers to a definite specific mineral, but it rather is a term which is applied to a variety of several different substances occurring as - in the fibrous form, and one could mention chrysotile and various other types of asbestos in accordance with a commercial term which has been given to them.

Now, the total number of persons engaged in the asbestos industry probably does not exceed 1,500. The number of such, at least in this country. The numbers of such workers inhaling asbestos dusts is not definitely known. However, industrial hygiene surveys and the frequency of pulmonary changes in such workers would indicate that only a few of the total number of men exposed to sufficient

quantities of asbestos fibers develop asbestosis. Thus, the condition differs considerably from the condition resulting from the present deposition of free crystalline silica. Certainly, it may go differently, the magnitude of the problem in one is different than in the other.

Now, it would appear that industrial environments that exist today necessitate generally ten or more years of exposure to asbestos dust in concentrations exceeding one million fibers longer than ten microns per cubic foot of air. It appears that way. This is basically subject to criticism and we know of other levels of permissible concentration that have been established for asbestos, but from our studies and our interpretation, and I repeat, it appears that industrial environments which exist today necessitate generally ten or more years of exposure to asbestos dust in concentrations exceeding one million fibers longer than ten microns per cubic foot of air.

Now, of course, exceptions to this occur. In subjects exposed to concentrations above that tentative permissible limit, and I recall to your mind, the statement brought out by Doctor Fletcher this morning, namely, the concentration. After all, if the concentration is high, well, then we find within the pulmonary tissue, many changes; so, too, with asbestos.

Now, an occasional case has come to us for study

with manifest pulmonary changes and profound symptoms, whose occupational history discloses an exposure of less than five years, but I again hasten to add in those instances, to an extremely high atmospheric concentration of long fiber asbestos. In view of the experimental studies, the pulmonary deposition of long fibers of asbestos seems to be the agent responsible for changes in the lung which are identified as asbestosis.

Rather than to cite all the experimental evidence, our evidence here at the Saranac Laboratory, and those by Smith, Woodin and King in England, have failed to show evident pulmonary change with fibers less than ten microns in length in our instance, and less than 2.5 microns in the instance of King and his workers, co-workers in England, and that, Whereas long asbestos fibers, in our experience, caused well-marked para-bronchiolar and interstitial resemblance, the condition, as we see it in human subjects exposed to asbestos.

Now, as you all know, the pulmonary changes due to the asbestos fiber occur initially as collars of fibrous tissue about the respiratory bronchials, and the progression of that fibrosis occurs and it is characterized by extension into the adjoining alveolar walls, which distorts those walls and distorts the formed alveolar space, giving rise often to what we identify as anatomical emphysema and

also to clinical manifestations of respiratory difficulty due, perhaps, to that emphysema and perhaps due to a diffusion phenomena. I shall not dwell upon that, because that is going to be a subject for discussion by Doctor Gregoire on Thursday.

The point to be made, however, is this, that asbestos manifests itself in an entirely different way than does asbestosis manifest itself in an entirely different way than does silicosis. Asbestosis is a diffuse pulmonary involvement, giving rise to the ground glass appearance in the lower lung, often involving the lower lung, manifested Roentgenographically. One condition, in our belief, is accompanied in many instances by respiratory difficulty, whereas the other condition, simple nodular silicosis, is often free of respiratory difficulty.

The action of inhaling asbestos fibers, I think the concensus of opinion today is, that it is a mechanical action, that the fiber being flexible, is inhaled into the respiratory bronchials and because of its flexibility, it can be taken up by a cell and it can be carried into the alveolar wall where it is deposited and produces its damage. Our experimental evidence seems to prove that since fibers which are brittle and just as long, glass wool fibers, which can be broken very readily, fail to produce this type of reaction which we see characterizes the pulmonary response

to the asbestos fiber. Thus we believe that the flexibility of the fiber is an important factor in the capacity of the asbestos fiber to produce damage in the lung.

There are some, however, who believe that the chemical actions, that asbestos produces its effect by chemical action. Supposing that the mineralogical coating of mineral substances is removed, leaving bare the asbestos fiber or spicule, allowing silica - silicic acid to be liberated and thus producing damage. I think that that needs proof. I don't - I do not believe that silicic acid produces the damage. We have been unable to reproduce the experimental effect, or reproduce it experimentally.

The asbestos bodies -- sometimes I like to refer to it as commonly called, body - asbestosis bodies, as the asbestos body. Why? Because these bodies appear anywhere in the lung. They often lie free in the alveolar space. The body is coated with a layer of substance which gives a positive reaction to iron, which we and others believe is derived from tissue fluids. I like to look upon the asbestos body as a - a body which has been removed or is incapable of producing damage. This has been cited by other workers, notably, Colus, Lynch and others, and I like to refer to it as the asbestos body rather than the asbestosis body, because the presence of this body does not mean that asbestotic fibrosis is present in the lung.

We have seen asbestosis, asbestos bodies appear in the sputum, without evident change in the pulmonary tissue. Therefore, the asbestos body in the sputum is not diagnostic for the disease asbestosis. It merely means that the individual has inhaled an asbestos fiber and that the body has reacted thereto by coating it with iron.

I have seen asbestos bodies lying free in the alveolar space, without evident pulmonary change in the alveolar walls which form that space. I have seen asbestos bodies in the alveolar wall without evident fibrosis. So, therefore, I like to look upon the asbestos body as a body which is incapable of producing damage, that their presence means merely that the fiber has been deposited in the lung, and that - and that their presence in the sputum has no reference whatsoever to the degree or extent of fibrosis in the pulmonary area.

Asbestosis is productive of pulmonary disfunction and this, too, shall be a topic for discussion on Thursday. Suffice it to say for the present time that the symptomatology of asbestosis established as bestosis as dominated by phthismia and irritating cough. The complications of asbestos, inhalation of the asbestos dust, does not apparently alter significantly, the final outcome of tuberculosis, either experimentally or clinically.

I know that there are those of you who will take

exception to that statement. However, our clinic study of clinical patients, our study of experimental animals, infected with the tubercle bacillus, have failed to demonstrate that the asbestos fiber deposited in the lung increases the susceptibility of that tissue to infection by the tubercle bacillus, thus asbestosis is quite different then or from silicosis.

Inhalation of asbestos dust and carcinoma of the lung is also a subject for discussion on Wednesday and I shall not dwell upon that at this moment. There is often associated with asbestosis, well-established asbestosis, cardiac enlargement that seems to result from increased pulmonary tension.

Bronchiectasis, identified as the dry type, is frequently associated as a complication of asbestosis, and it is logical since the fibrosis of asbestosis gains about the respiratory bronchioles and about the bronchials, that there is a degree of fibrosis which interferes with the wall of the respiratory bronchiole and which will often cause a physiological disfunction, thus retention of secretions, thus also the appearance of bronchiectasis.

Experimental evidence with respect to asbestosis indicates, and also there is some clinical evidence, that on removal of the asbestotic from the - from the atmosphere, that there is appreciable clearing of the asbestosis of the

lesion in the lung, that, thus, it also differs from silicosis. Remove a man with silicosis from his - the atmosphere containing free crystalline silica, that silicosis will progress for a short period of time and then it will become stabilized and indeed, it may regress by reason of contraction of the fibrous tissue, but with the asbestotic, that individual, on removal from the dusty atmosphere, the disease in his lungs seems to stabilize very quickly, much more quickly than that does to the free crystalline silica particle.

As far as therapy is concerned, studies by the Saranac Laboratory and also by Smith, Woodin and King, experimental studies with aluminum hydroxide, colloidal aluminum hydroxide, showed no evident retention or retardation of the development of asbestosis in experimental animals. Thus, again, it differs from the reaction due to the free crystalline silica in the form of quartz.

Furthermore, this asbestosis, this aluminum hydroxide or aluminum, colloidal aluminum hydroxide did not prevent the development of the reaction to the asbestos fibers deposited in the lung. If anything, our experimental evidence, and that is also supported by the evidence from King in England and his co-workers, is that aluminum hydroxide augments the fibrosis due to the asbestos fiber.

Cortisone? I know of no experimental or clinical

study where Cortisone has been used as a possible therapeutic agent against the pulmonary reaction to asbestosis, to asbestotic fiber or for asbestosis. It seems to me, however, that Cortisone should be given a trial. We have considered that for silicosis, but by reason of the fact that the individual with free crystalline silica in his lung is more susceptible to tuberculosis, we have not favored the use of Cortisone which also increases the susceptibility of the lung to tuberculosis. But that does not appear to be the case in asbestosis.

We, therefore, consider giving Cortisone a trial in the asbestotic. Thank you.

BY DOCTOR HATCH:

I'm a little curious to know why, in settling the times for this afternoon, Doctor Vorwald has allotted more time for discussion of Beryllium. I thought Doctor Hardy could tell us all about it in five minutes, but instead of that, she is allotted a little longer time. I'll ask Doctor Hardy to talk on Beryllium.

BY DOCTOR HARDY:

Professor Hatch and Members of the Symposium:
There has been in the past decade a truly impressive accumulation of American and foreign literature relating to the epidemiological, clinical and experimental aspects of exposure

Corrected
By
DR Hardy

to certain beryllium compounds. This documentation and active attempts to use this data in the prevention of worker illness reflect the accuracy of Dr. Alice Hamilton's⁽¹²⁾ observation that in her experience she has never seen a more prompt and active attack on a serious industrial hazard. Much of the available knowledge of beryllium intoxication is well known to the membership of this Symposium. Because of this and the size of the subject, I shall in the time at my disposal deliberately choose certain current observations and opinions which seem to me to warrant your attention. I shall present this material under the headings of the epidemiology, the clinical aspects, the treatment and the diagnosis of beryllium poisoning.

Epidemiology:Table I

~~This Slide I and the one following (Slide II) represent~~^s the operations which I know with reasonable certainty have been incriminated as causing worker illness. And ~~this table~~^{II} ~~(Slide III)~~ shows the compounds associated with these operations. Van Ordstrand⁽²⁸⁾, Wilson⁽³⁵⁾, DeNardi⁽⁵⁾, and I agree that beryllium oxide appears as the usual etiologic exposure in the Be. ~~Indus.~~^{Beryllium Poisoning.} illness in the extraction industry both here⁽³³⁾ and abroad^(26,30) is held to be associated with exposure to the acid salts of beryllium especially the fluoride and the sulfate. Accurate information on incidence of cases of beryllium poisoning associated with certain work

exposures is difficult to obtain. Lack of knowledge of total number of those exposed, failure to recognize beryllium-caused disease in the past and continued appearance of cases of chronic poisoning long periods after cessation of exposure combine to make available information of case incidence undependable. Here are a few reasonably accurate figures. (5) DeNardi and associates report 431 cases of acute beryllium reaction in an extraction plant between 1940-1952 during which time some 1,700 people were employed. 222 of such illnesses involved the respiratory tract with 60 cases of acute beryllium pneumonitis, 10 of which proved fatal. Vigliani reported in 1948, (30) 75 cases of bronchitis among 230 workers engaged in alloy manufacture, all disability being transient. (26) Tabershaw, during a trip to Germany in 1950, learned from Niemoeller that in 1936 of 112 workers exposed 43 became ill following exposure to beryllium salts, 3 dying during an acute reaction and 3 at an unnamed later period. It is apparent from such evidence that acute beryllium poisoning is related to intensity of exposure. Absence of new cases of acute disease as reported to me by Eisenbud, (9) following control of working air concentrations of beryllium to below 25 micrograms/m³ of air for the briefest exposure is further proof of the relation between beryllium concentration and disease.

The problem of case incidence in industrial

epidemics of chronic beryllium poisoning is far from settled as clinically active cases are currently appearing from beryllium exposures which ceased as long as 10 years ago. Here are a few recently acquired figures for your consideration. In one fluorescent lamp manufacturing plant between 1,400 and 1,700 people were exposed to a phosphor containing about 12% beryllium and to date there are at least 90 diagnosed cases of chronic beryllium poisoning. This is an incidence of over 5% which is greater than the 4% considered epidemic for a communicable disease in a community. In fluorescent lamp manufacturing plants using a less than 4% beryllium containing phosphor a small number of cases of chronic disease have been documented - roughly 15 of several thousand exposed workers. This experience indicates that both high and low beryllium-containing phosphors can cause disease. Since high and low beryllium-containing phosphors were prepared differently with low and high firing⁽³²⁾, there was variation in the physical characteristics of the beryllium compounds.

⁽⁵⁾ DeNardi[^] reports the low figure of 7 cases of chronic beryllium disease from the roughly 1,700 workers on extraction operations among whom 431 cases of acute beryllium reaction occurred. Relatively high incidence of⁽²⁴⁾ chronic beryllium disease has been reported by Slavin[^] from spraying of cathode tubes and by Saunders⁽²³⁾ in a small neon

sign manufacturing shop where phosphor preparation and tube coating were done. There are many gaps in our knowledge of the epidemiology of beryllium disease in alloy manufacture, Beryllium research⁽¹³⁾, certain operations in neon sign manufacture⁽¹³⁾, neighborhood contamination⁽³⁾ and beryllium metallurgy. No cases of beryllium poisoning have been reported from handling beryl ore which, if correct, I believe means that qualitatively beryl will not produce human beryllium disease. Finished beryllium alloys containing less than 4% beryllium have not caused illness to date, which may be interpreted to mean that quantitatively there probably is a safe level of beryllium air contamination. Some of our gaps in knowledge will never be filled, I am glad to say, because proper engineering control will prevent new cases and we lack data on beryllium air contamination of past operations.

Scanty though it is, knowledge of epidemiology has led to acceptance of beryllium as etiology and to attempts to explain the mechanism of beryllium intoxication. Sterner⁽²⁵⁾ has studied the available data and presents the broad concept that clinically active beryllium poisoning is an immunotoxic reaction to the chemical held in tissue. Curtis⁽⁴⁾ backs this up by describing positive skin tests in all cases of active disease. These observers, if I quote them correctly, consider that some inherent reactivity of the individual is decisive in producing disability. It seems to me that this

may be true and if it is, the same mechanism rules the appearance of clinically active infantile paralysis and other infectious disease when such disease is epidemic in a community. Yet, it is seen that the high incidence of chronic beryllium poisoning reported by Slavin⁽²⁴⁾ in a small group spraying radio tube cathodes and the variation in case incidence between high and low beryllium phosphor fluorescent lamp manufacture are examples of the importance of quality and/or quantity of beryllium exposure in chronic as well as acute disease production. The delay between cessation of beryllium exposure and onset of clinically active disease leads me to believe that we are still faced with understanding what Gardner called 'beryllium plus the x factor' which I, as a clinician, have described as the precipitating factor. In summary, then, sketchy as our knowledge is, the epidemiology of clinical illness clearly correlated with certain beryllium operations has taught us several facts. These facts are that some acid salts of beryllium and various forms of the oxide have caused poisoning, acute and chronic, among workers and a few individuals living near beryllium operations. Reduction of beryllium concentration in working environment below $25\text{M}/\text{M}^3$ ^{microgram} for short and $2\text{M}/\text{M}^2$ ³ for ^{steady} high exposure has prevented the development of acute beryllium disease in the past 4 years. Because of the delay in onset of the chronic disease we do not yet know if these levels are

low enough to prevent chronic beryllium poisoning.

Clinical Aspects:

The published work of Van Ordstrand⁽²⁹⁾, DeNardi⁽⁵⁾, and associates makes it unnecessary for me to describe acute beryllium poisoning. Their observations lead to certain conclusions of importance in the study of beryllium disease. According to Van Ordstrand, acute beryllium poisoning when manifested as a pneumonitis is a constitutional disease giving weight loss and biochemical changes as does the chronic disease. DeNardi in a recent report makes the interesting point that workers in the extraction industry exposed to acid beryllium salts exhibit higher concentrations of beryllium in urine and tissue than do workers in industries such as fluorescent lamp manufacture studied by Klemper⁽¹⁵⁾. This finding occurs with and without evidence of clinically active disease and after cessation of beryllium exposure. Kelmer² has found that Be₂ is not found in the urine of non-exposed controls. I interpret this to mean that while intensity of exposure determines clinical acute beryllium reaction, the body is capable, to a remarkable degree, of handling beryllium safely by excretion and storage. Further, I conclude that quantities of beryllium in urine are no index of disease.

Table III

~~This slide IV~~ lists my arbitrary description of clinical syndromes associated with chronic beryllium

poisoning. I want to make certain points by describing a case in each category.

Here is the chest X-ray ~~(slide V)~~ of C. G. who worked as an engineer in the development of fluorescent lamp manufacture for 4 years, leaving 10 years ago. During an attack of infectious hepatitis 9 years ago this chest X-ray was discovered. He has never missed a day's work, admits to no symptoms and has the same X-ray picture this year. As we gain experience and with more chest X-ray surveys we see a definite number of such cases. I refer to this as asymptomatic beryllium effect.

L. S. ~~(slide VI)~~ is a 36-year old engineer who exposed to high beryllium phosphor for 3 to 4 years during his work designing and perfecting the metal frames for fluorescent lamps. He noted raised red skin lesions on his face and anterior chest wall 4 years ago. These were variously treated with dessication, chemicals, radiation, and excision without diagnosis. In 1949 after a mountain climbing trip this man caught cold. He did not recover as he thought he should and noticed dyspnea on effort^{out} for the first time. The X-ray you see here is similar to the one taken at the onset of symptoms. Tuberculosis was carefully ruled out. Biopsy of the skin lesions revealed a granulomatous reaction very like sarcoidosis. This man does a sedentary job, tires easily, has dyspnea on exertion such as walking up a slope.

As in the case of the first category, in our experience at least, this group of cases of mildly disabling beryllium disease is growing.

A small group of cases of chronic beryllium poisoning exhibit dramatic febrile episodes that cannot be controlled with any drug. ~~This (Slide VII) is a slide of The~~
~~case of A.R., showing the chest X-ray changes of such a pa-~~
~~tient in such an episode.~~ In my experience such episodes reflect a poor prognosis. Can it be that the febrile reaction means a mobilization of beryllium from its body storehouse?

This X-ray ~~(Slide VIII)~~ belongs to B. L., a young woman exposed to beryllium oxide in ceramic work. She was probably quite heavily exposed while shaking the dry material for sizing purposes a part of each work day for 18 months during 1944-45. Her symptoms began in 1950 with hacking cough and later dyspnea. She has had to give up even desk work but has never been a bed patient, maintains good appetite and steady weight. Such patients in my classification are moderately disabled.

An interesting and controversial group is represented by the following X-rays of W. ~~X.~~^{A.R.} ~~(Slide IX)~~. This man worked in the preparation of phosphors containing about 12% beryllium for use in fluorescent lamp manufacture. He had acute beryllium pneumonitis in 1943 as shown here with

complete symptomatic recovery. In 1947 he became disabled with cough, dyspnea and weight loss. Here is his later X-ray. ~~(Slide XX)~~. After a long course W. R. died of chronic beryllium poison. ^{I have} ~~my~~ first hand knowledge ^{of} extends ^(5a) to 7 such cases. It is of interest that DeNardi ^N reports that 5 of his cases of occupational chronic beryllium poisoning had suffered acute beryllium tracheo bronchitis at an earlier date. He also reports 1 case of acute beryllium pneumonitis - the active phase occurring in 1944 - which has never recovered and now shows X-ray evidence of pulmonary fibrosis. This finding of a few chronic cases after acute beryllium reaction raises the important question of whether a worker who has shown evidence of acute beryllium poisoning of the respiratory tract should be allowed to return to beryllium exposure. If Sterner's concept is correct, such workers have demonstrated a harmful reaction to beryllium in any dose, or perhaps one might simply say insult should not be added to injury.

~~(Slide XIX)~~. There are a number of cases of chronic beryllium poisoning with bad prognosis, like that of A.K. shown here who are completely disabled. These patients may have lived as long as 8 years in my experience dependant on nearly total rest with oxygen handy ~~(Slide XX)~~. ~~They are cachectic as this slide of R. G. illustrates.~~ From case to case, at intervals can be demonstrated evidence of negative

nitrogen balance, liver involvement, splenic enlargement, ~~increase in~~ ⁱⁿ total protein, gamma globulin, altered alkaline phosphatase. Renal calculi occur in about 20% of all cases of the ~~(Slide XIII)~~ disease with and without hypercalcuria or hypercalcemia - often causing little disability. ~~In this case of this fluorescent lamp worker, ill for 9 years, the stones were so large and she so thin, they permeated anteriorly through the abdominal wall. There was evidence of involvement of lung, liver and spleen at the same time. As expected cor pulmonale develops if the patient lives long in this state. The mortality rate is at present 20-25% and my data shows complete invalidism of 50-60% of the cases. You will recall that I listed recovery as a possible clinical outcome of chronic beryllium poisoning but put the word in quotation marks. This was done because I know of no case in which the patient has become symptom and sign free after a serious episode of illness.~~ ~~This (Slide XIV) is the chest X-ray of~~ (2) ~~represents a man who~~ ^{represents a man who} F. L. whose case is reported in full by Cass. ~~This man had~~ been exposed to high Be. phosphor. He was desperately ill for more than a year. Now, 7 years after sufficient recovery to go back to his desk job he still shows fine X-ray densities and evidence of right heart strain. He has passed small renal calculi on 3 occasions and has at intervals brief bouts of unexplained chills and fever from which he spontaneously recovers.

omit
OK-H.H. →

Change all references
Slide XIII

Although I have spoken only briefly of each category, I hope you sense the protean manifestations of toxic beryllium effect. It is my opinion that these various clinical pictures, the many biochemical and postmortem changes, as well as finding beryllium so widely distributed in bone and tissue indicate that beryllium poisoning is a constitutional disease. Van Ordstrand⁽¹⁴⁾ and DeNardi⁽⁵⁾ agree with this but Wilson⁽³⁴⁾ and doubtless others do not and prefer to consider this disease purely pulmonary.

I have wondered if increase in the period of delay in onset between cessation of beryllium exposure and clinically active disease would change the prognosis. De Nardi⁽⁵⁾ thinks not from his experience. However, the new cases of chronic beryllium poisoning I see now are not showing the malignant course of those I first saw in 1945-47 from fluorescent lamp manufacture using 12% beryllium phosphor. This may be because at least some of the cases I see now are from different beryllium exposures, different both qualitatively and quantitatively.

It is of practical clinical importance to know that there have been a negligible number of acid fast infections complicating chronic beryllium poisoning. I know of only 3 such cases. Further, although animal experimentation shows that Gardner's⁽¹⁶⁾ finding of osteogenic sarcoma in beryllium exposed rabbits can be successfully repeated⁽¹⁷⁾, there has been

no clinical experience to incriminate ^{beryllium}beryllium as a carcinogen. To my knowledge there has been only 1 case which clinically appeared to be chronic beryllium disease and at autopsy proved to be pulmonary adeno-carcinoma. No characteristic histopathology of the chronic beryllium reaction was described. Minute but measurable amounts of beryllium were detected in the lung. In one other instance reported by ⁽⁵⁾DeNardi, beryllium was found in postmortem tissue of an exposed worker whose lungs showed bronchiogenic carcinoma. This man had had acute beryllium pneumonia. These reports are of interest but certainly not valid at this time in relating beryllium exposure to malignant disease.

I shall speak briefly of the treatment of chronic beryllium poisoning. ACTH and cortisone have been shown to affect the disease to a remarkable degree. Pulmonary function study ^(9, 20, 31, 32)demonstrates real change in the underlying process in many but not all cases studied. Biopsy study before and after treatment has been reported as both showing no change and decrease in the ^{number}number of characteristic granulomata ⁽¹⁷⁾. In many cases the X-ray densities of chronic beryllium poisoning have shown definite lightening with Rx. In my experience the X-ray picture may progress unfavorably as shown by emphysema and increase in the size and number of the densities in spite of treatment. ⁽¹⁵⁾Klemperer has satisfied himself that cortisone does not increase the excretion of

beryllium. The action of ACTH and cortisone on other systemic effects of beryllium is interesting and mystifying. Enlarged liver, spleen, and hilar nodes may be reduced to normal size; disturbed liver function as reflected in bromsulfalein retention may be reversed. Increased total protein, gamma globulin, change in alkaline phosphatase levels are restored to normal values by treatment with ACTH or cortisone. Recent experience has given me evidence, on the other hand, of the relentless progress of chronic beryllium poisoning in some cases in spite of a relatively symptom free status of the patient following the use of these drugs. In one carefully followed case emphysema has developed in spite of treatment; in another, abnormal liver function tests and increase in X-ray visible pathology of the chest in spite of improved pulmonary function values and few symptoms, suggest that we do not have a cure for beryllium poisoning in using either ACTH or cortisone.

My experience leads me to state, however, that clinical improvement of symptomatic chronic beryllium disease is so spectacular that one is not justified in withholding these drugs. There have been few untoward side effects in these cases known to me. Transient glycosuria, gastritis, and one questionable psychiatric reaction make up my experience and do not seem worrisome in view of the poor prognosis of this disease. From intermittent courses

courses of ACTH, I have changed to short high level courses of ACTH followed by long term use of oral cortisone in dosages which will control dyspnea and cough. Differing from Van Ordstrand⁽²⁸⁾ and DeNardi⁽⁵⁾ et al, I and my colleagues, Wilson⁽³⁵⁾, Hasterlik⁽¹³⁾ and Kline⁽¹⁷⁾ and, I am sure, others use these drugs in certain cases of chronic beryllium poisoning that are less than severely disabled. In a few chosen cases, I have treated workers with only X-ray changes and perhaps dyspnea on strenuous exercise where the diagnosis has been made on epidemiological grounds and beryllium has been found in the urine. It is too early to know whether or not this approach is worthwhile. In summary, it is my current belief that either ACTH or cortisone or both used in dosages to control symptoms for long term courses varied to suit the individual case are of real merit in the treatment of chronic beryllium poisoning. I do not believe these drugs are curing the disease but they are providing at least some patients with means for more efficient handling of the toxic insult.

^(23a) There has been a good deal of interest in Schubert's^{A.T.A.} use of ^(aminotris carboxylic acid) Aurin^A to improve the survival time of animals^(9a) acutely poisoned with certain beryllium compounds. Finkel^{A.T.A.} has shown that sodium salicylate chemically allied to Aurin^A will also provide^{Some} protection to beryllium poisoned animals. ~~Aurin provides much greater protection than the salicylate.~~
A.T.A.

Work with chronically poisoned animals such as those of Dutra⁽¹²⁾ showing granulomatous skin reaction to beryllium and toxicity ^{A.T.A.} studies on Aurin are waited with great interest. If this chemical should prove the specific for the toxic beryllium ion as Schubert postulates, it may be as speculated that ACTH or cortisone might have an effect on the granulomatous reaction of chronic beryllium disease after which ^{A.T.A.} ~~Aurin~~ could act.

Diagnosis of Beryllium Poisoning:

During the time remaining I want to talk to you about the diagnosis of beryllium poisoning. The acute disease does not warrant discussion because its occurrence is reasonably well correlated with beryllium exposure of relatively high intensity. It also occurs either immediately or within a matter of a week of such exposure.

The chronic disease, because of delay in onset and because of what often appear to be very slight causative beryllium exposures, presents a much more complex problem. Machle⁽¹⁴⁾ and Williams⁽³²⁾ have pointed out that knowledge of the physical characteristics of certain beryllium compounds causing illness may make the appearance of cases less mysterious. Also, as I said earlier many facts suggest that beryllium may act with another factor to produce disability. Dr. Gardner called this the x factor, Dr. Sterner⁽²⁵⁾ calls this an immunotoxic reaction of a susceptible individual. I

for example [§] have referred to it as the precipitating factor which may, ~~for example~~ be an antecedent respiratory tract insult, bacterial or chemical, post pregnancy endocrine activity, or induced weight loss.

(34), (12)
Wilson, Roberts, and others studying the chest X-ray changes of chronic beryllium poisoning have taught us correctly that lymphatic spread of malignancy, acid fast infections, sarcoidosis must be considered in each case. Proper studies will usually settle the diagnosis in the first two cases. Sarcoidosis involving the lung has become our great diagnostic problem as we see the wider spectrum of clinical syndromes following beryllium exposure which I described to you earlier.

~~Table IV~~ 4
In the next two slides are summarized the features that chronic beryllium disease and sarcoidosis have in common. (Slide XV).

The chest X-ray changes in both beryllium poisoning and sarcoidosis are certainly bilateral and with rare exceptions involve the entire lung fields. The hilar node enlargement and spontaneous pneumothorax secondary to large emphysematous bullae are common to the two diseases. Greater use of routine chest X-ray study has substantiated the truth of the poor correlation between clinical complaint and X-ray densities in both diseases. The occurrence of renal calculi ⁱⁿ chronic beryllium poisoning is unexplained as in

sarcoidosis where calculi also occur though less frequently.

~~(Slide XRT)~~. It has been of great interest and disappointment that in the studies thus far completed biochemical, physiological and histopathological studies do not give completely clear and helpful differential points between beryllium poisoning and sarcoidosis. To illustrate the kind of problems currently being encountered I would like to speak of P. K., a 32 year old Navy veteran who prior to entering military service worked as inventory clerk near salvage operations of fluorescent lamps made ^{with} beryllium containing phosphors. In December 1950 because of hacking cough with fine widely scattered X-ray densities in the chest and otherwise negative examination a diagnosis of sarcoidosis was made. His family physician ruled out pulmonary tuberculosis and discovering his exposure to beryllium referred him to us. We found beryllium in the urine, beryllium in lung tissue and slightly abnormal pulmonary function values. The lung seen during thorocotomy for biopsy showed widespread, greyish firm nodules which on microscopic study were variously called sarcoidosis and chronic beryllium poisoning by different observers. It has seemed to me reasonable to conclude in this man's case that what we found may correctly be correlated with beryllium exposure.

Experience is, of course, still gathering on reaction to ACTH and cortisone in both beryllium poisoning and

sarcoidosis. When I made this ^{table} ~~slide~~ my experience and that of others pointed to favorable response of some degree in both diseases. Since then I have seen cases of sarcoidosis unresponsive to these drugs. There is, of course, the possibility that the same will prove true with beryllium disease, but as yet we have only favorable reports in, except in the few cases where we are trying preventive therapy impossible as yet to evaluate.

To try to bring out the diagnostic differences I now want to go through ^{table V} ~~two slides~~ (Slide XVII) of listing the variation between sarcoidosis and chronic beryllium poisoning. I have been able to crystallize these differences by study of such material presented by Longcope and Freiman⁽¹⁸⁾ and the collection of cases from the military made by Michel⁽¹⁰²⁾ in Georgia. The clinical findings are placed in the order in which they seemed most striking in comparing the two diseases. You will recall that our few remissions of chronic beryllium disease continue to show X-ray changes and varying amounts of disability much as a case of treated tuberculosis, not a true recovery as ^{may occur} in sarcoidosis. The only change in the hemogram of chronic beryllium poisoning known to me is the secondary polycythemia^C. In sarcoidosis there is usually increase in the total white count, made up of monocytes, and in some series eosinophiles. Only an occasional case of beryllium disease shows an increase in

sedimentation time. You will notice that I have not recorded the changes in the tuberculin tests. The fact is I do not have in the chronic beryllium poisoning cases good data on this point which is valid for comparison with the oft reported negative tests in sarcoidosis. The impression I gain from my own experience is that carried to strong enough concentrations the tuberculin test in our series of cases of beryllium poisoning is about what it would be in the general population.

It is of importance that no X-ray changes have been seen in the bones of cases of chronic beryllium poisoning whereas in 20-25% of the cases of sarcoidosis characteristic changes are reported. This is important not only because it affords a differential diagnostic point but because of the small-animals study showing that beryllium compounds can produce osteosclerosis and in ^{rabbits} ~~certain animals~~ osteogenic sarcoma. During a recent lung biopsy for diagnosis of respiratory disability in a beryllium exposed worker, a rib was removed. We were surprised to learn that although a definite but small amount of beryllium was present in the lung, over 100 times as much was found in the rib. The man's last beryllium exposure was 10 years prior to the biopsy. Furthermore, 2 autopsied cases - one to be reported in full by Brown - have shown a curious hardness of the bone, referred to informally as "marble bones." One cannot help but

wonder if workers living for many years with stored beryllium of possible pathogenicity may not develop clinically detectable bone disease.

(15), (5),
 My knowledge of the work of Klemperer⁽¹⁵⁾, DeNardi⁽⁵⁾,
 Dutra⁽⁷⁾ and Cholak^(3a) leads me to use the presence of beryllium in biological material determined chemically or spectrographically as signifying exposure only and not intoxication. As in the diagnosis of lead poisoning, the discovery of beryllium in the urine of a case under study is only one part of the evidence needed in establishing etiology, although an important one if the test is done in a reliable laboratory.

~~(Slide 8 VIII)~~ In the past I have been much impressed with the greater morbidity and mortality of chronic beryllium poisoning over that of sarcoidosis. However, my own experience and Reisner's^(21a) work bring the two diseases together on this point also. Reisner reports 20-25% mortality for sarcoidosis which is very near that of chronic beryllium poisoning in most reported series. It is true that the gastrointestinal symptoms and great weight loss in some cases of chronic beryllium poisoning are in striking contrast^{ing} to sarcoidosis. This may be a reflection of inability to absorb and store nitrogen shown in the metabolic studies of Waterhouse⁽³¹⁾.

The absence of eye, parotid and tonsil involvement

in chronic beryllium poisoning, I put at the end of the list. We have never seen these but as we constantly gain new knowledge of the clinical possibilities of this disease we may encounter changes in these tissues at a later date.

Let me say in summary that in my opinion the correct diagnosis of beryllium poisoning rests on the establishment of exposure by epidemiological or environmental study or lacking these, its discovery in urine, blood or tissue. In addition clinical judgment and imagination in evaluation of symptoms, laboratory studies, X-ray opinion, and physical findings are required as experience with beryllium intoxication broadens the spectrum of syndromes that may occur.

We know that beryllium and its compounds will continue to be used and the evidence is complete that certain compounds produce disease after a delay of uncertain length. May I therefore urge conscientious epidemiological studies of beryllium exposures and illness when it occurs, studies of physical characteristics of the beryllium compounds in use, and acceptance of the protean manifestations of beryllium effect in the body.

~~(Applause)~~

BY DOCTOR HATCH:

Doctor Hardy is certainly to be congratulated for covering so much on beryllium in the time allotted to her. I'm sure that many of you will have questions to raise with

H#1

VORWALD COIL

BOX 89

34pp

Discussion

3

DR. MACHLE: I'd like to compliment Dr. Hardy on a very excellent summary of the beryllium problem and to ask her to elaborate on the cases presumably exposed to low-beryllium phosphor. We have been following data on several thousand persons exposed since 1945 to low-beryllium phosphors and have yet to find a case of the beryllium disease in that group.

3
is a mixture of knowledge that I have by the courtesy of other physicians.
DR. HARDY: My data ~~includes material supplied by another physician, which~~
~~refers to a phosphor having, I understand, a beryllium content always below~~
~~4 per cent..~~ He reports about 11 cases and we have in Massachusetts 2 cases
in or from companies using ^{a low beryllium} ~~that same~~ phosphor. I have ^{a report} from Nash of England,
^{reporting} ~~reports~~ of 6 cases of chronic beryllium poisoning, 3 of these from low-beryllium phosphor manufacturing. I used the word "roughly" in my paper
advisedly because I ^{do not} ~~didn't~~ have ^{the} precise data as to the total number of cases, but it
^{is something of the order of at least} ~~There were~~ about 15 cases, - or possibly more - out of ^{These cases are from} an unknown number,

in data not checked by Tolson
OK
Machle

OK
in data not checked by Tolson
Machle

HHZ

low beryllium

doubtless in the thousands, of individuals exposed to phosphor. ~~with~~
beryllium content of less than 4 per cent.

*not beryllium
not phosphor
any more*

*OK
Machle*

DR. MACHLE: That is a very significant observation. There are several variables in beryllium phosphors which have a bearing on exposure. One is the total amount of beryllium present, since there is a limit to the amount that will react with other components of the phosphor. A second variable is the completeness of the reaction which, of course, is affected by the time, temperature and repetition of firing and by the use or omission of fluxes. In at least two situations involving cases which seemingly occurred from exposure to low-beryllium phosphor only, we have been able to show that there were unusual practices in the preparation of the phosphor - lessening the firing, experimenting with fluxing methods, and so forth - which lead one to believe that those situations were anomalous and should not be included in the general epidemiological consideration.

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Dr. Hardy corrected

³ UNIDENTIFIED SPEAKER: Dr. Hardy, what is the longest period of time for treatment of those patients to whom you have referred?

OK

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DR. HARDY: One patient, ~~recently~~ reported by Kennedy in the ~~J.A.M.A.~~ ^{J. Canad. Med. Assoc.}, I ~~have continued to treat up to the present time.~~ ^{has been treated intermittently since January 1950.}

UNIDENTIFIED SPEAKER: One of your patients looks like a hyperthyroid. Is there any indication that toxicity stimulates the thyroid, or is hyperacidity of the bone the cause?

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~~that.~~ We had two cases of thyroid tumors which seemed to disappear. They had high B.M.R.'s ~~and hypercalcemia.~~ *by biopsy* Their lungs were not explored to learn what had happened. We feel that in some mysterious way the disposition and modus operandi of beryllium in the body must be related *to the handling of calcium* but at present I have no information on the subject except the early tracer studies of Dr. Hamilton Berkeley in which beryllium put into animals remained in the liver and kidney for a while but never left the bones for the duration of his study. This situation is in agreement with the findings in autopsy cases when beryllium was present. Dr. Fuller Albright believes that the renal calculi and perhaps the other phenomena I enumerated are related, just as he believes they are related, in sarcoidosis, to a protein binding - in certain diseases protein will bind calcium. But that is really a description, not an explanation.

UNIDENTIFIED SPEAKER: What is the dosage of ACTH you used for treatment?

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 DR. HARDY: It varies a great deal. In general we have given 200 milligrams *intramuscularly* *every 6 hours for a few days and later the milligram doses;* of ACTH a day, divided into 50 milligram doses ~~spread out~~; then a shift to oral cortisone. The patient is kept on that treatment for a week or two until we're sure we have what would be the best possible benefit from the symptomatic standpoint. In various cases we have tested this by pulmonary function study and cardiac catheterization study. We then do what Dr. George *Thorn* Dorr calls "titrate the dose", meaning that we decrease the dose to the point *at* below which the patient is still comfortable, and then continue with that dose.

I have patients that have been on 75 milligrams of cortisone a day -
25 milligrams every eight hours - for a year and ^{we} ~~I can't~~ see ^{few harmful effects,} where I've

done any harm. I've changed symptomatic patients from being completely
chair patients to individuals able to be up four hours a day. This is

very impressive even though I don't believe that ^{I am} ~~I'm~~ curing them. ^{beryllium poisoning.}

UNIDENTIFIED SPEAKER: Dr. Kane of Michigan seems to think that the use
of cortisone may lead to sporadic sclerosis.

DR. HARDY: It might easily be true but I believe that in the case of
beryllium poisoning one must take that chance.

DR. VORWALD: Is that a general tendency with cortisone?

DR. HARDY: Yes. It has been my experience and the experience of Dr. Max
Michel, who has treated ^{cases of Boeck's sarcoid} ~~very many of these cases~~, that you may get ^{apparently} ~~bril-~~
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DR. TEBROCK: Our experience in the treatment of chronic beryllium poison-
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Dr. Hardy, what in your opinion is the activating factor in the precipitating mechanism? Is it a true allergy or is it a matter of physico-chemical changes within the system that precipitates the disease?

3.
DR. HARDY: I believe that the beryllium in the body is in a state of equilibrium, is stored for excretion, and then something happens to the individual, ^{such as} ~~Maybe it is~~ a respiratory tract infection or a pregnancy or, for soldiers, experience in combat or loss of weight incurred during boot training. In practically all cases in which I have been able to make a good clinical hunt I have found that something happened to the individual after he or she stopped inhaling beryllium or that the individual was subjected to an added physiological burden which made it possible for the beryllium to become pathogenic.

I have not seen any evidence that the clinically-active disease is an allergic type of response but I believe that Dr. Sterner has a good basic idea that may explain many things about ^{all} ~~geiter~~ response to toxic insult, not just to beryllium but to infection also.

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industrial worker, caused by beryllium, are as nearly similar to each other as are the lesions produced by silica in animals and in man. Lesions developed after many months of exposure, at an extremely low level of concentration, to pure beryllium oxide or to beryllium sulphate. We have not yet succeeded, for unknown reasons, in reproducing the number of lesions seen in human cases, as the lesions formed in the experimental animal are few and far between.

Discussion

DR. MACHLE: I/^{would} like to compliment Dr. Hardy on a very excellent summary of the beryllium problem and to ask her to elaborate on the cases presumably exposed to low-beryllium phosphor. We have been following data on several thousand persons exposed since 1945 to low-beryllium phosphors and have yet to find a case of the beryllium disease in that group.

DR. HARDY: My data is a mixture of knowledge that I have by the courtesy of other physicians. Dr. Edward Klein reports about 11 cases and we have in Massachusetts 2 cases in or from companies using a low-beryllium phosphor. I have a report from Nash of England, reporting 6 cases of chronic beryllium poisoning, 3 of these from low-beryllium phosphor manufacturing. I used the word "roughly" in my paper advisedly because I do not have the precise data as to the total number of cases, but it is something of the order of at least 15 cases. These cases are from an unknown number, doubtless in the thousands, of individuals exposed to low-beryllium phosphor.

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BY DOCTOR HATCH:

Gentlemen, to continue our discussion now on the newer problems, with a report by Doctor Pratt of the

Laboratory staff on their studies on some of the new synthetic silicas. Doctor Pratt.

BY DOCTOR PRATT:

(Doctor Pratt read a prepared paper, which is on file at the Saranac Laboratory).

BY DOCTOR HATCH:

Ladies and gentlemen, we have heard now about many different kinds of materials ranging all the way from our old friend Silica, as it occurs in nature, to problems that develop with the synthetic silicas, and these other curious dusts in between.

We go now to a little departure, perhaps, from the processes that have been followed this afternoon, the discussion of the behavior of particulate matter, without special reference to the nature of the material, in the course of inhalation and certainly the way in which particulates are handled in the course of inhalation and their receipt, and the amount of retention, and the understanding of those things is fundamental to the building up an understanding of the whole problem of dust diseases.

Merril Eisenbud of the New York Operations Office of the Atomic Energy Commission will report to us now on the Fate of Inhaled Particulates. Mr. Eisenbud.

BY MR. EISENBUD:

(Mr. Eisenbud read prepared paper which is on file

in the Saranac Laboratory).

BY DOCTOR HATCH:

I think you will all agree with me that we have had a very concentrated afternoon and I'm not sure how much time you're going to feel like taking now for discussion, but I am certain that whether you feel like it or not, you all have many questions that you'd like to raise for such discussion.

I should like to emphasize at this point, the chairman's privilege of opening the discussion and to make one or two comments which pertain particularly to the last presentation of Mr. Eisenbud's, and having to do with the fate of inhaled particulates. First of all, I'd like to suggest, whereas there has been a good deal of attention paid to initial retention of particulate matter in relation to particle size or the - over the last few years, I'm not at all sure in my own mind that it's the most important factor compared with the subsequent clearance of particulate matter from the entire lung structure. I remember how impressed I was many years ago when I made a very crude little calculation with some of the data from South Africa comparing the amounts of materials found in matching of silicotic lungs with some rough calculations on my own among the material that the individual had probably inhaled and retained, and while the essence is necessarily very crude, nevertheless

the order of magnitude was not far off, and the amount retained or the amount accounted for in the sillicotic lung was in the order of one tenth to one percent of the amount which they had inhaled over the years.

Now, then to whatever degree that residue represented the material that it produced in them, it's pretty clear that we're talking about a highly selected portion of the total material inhaled, and so when we try to relate the composition and the physical characteristics and so on, of the material in the atmosphere, among them it seems to me pretty evident, that there is an awful lot of information - an awful lot of things happen from time of inhalation to the development, full development of the effects, so I suspect that the - one of the biggest gaps in our understanding, one of the things in which there is the greatest need for research, is the research into the way particulate material is handled, the rates, relative rates of clearance and the relative ways in which the particulate matter is disposed of within the lungs and in that connection, I'd like to make two points which will amplify, add perhaps a little bit, to what Mr. Eisenbud has just had to say about the retention.

First of all, if you will permit me to make a very diagrammatic sketch here, of a primary molecule, and this is very diagrammatic, but I want to use this way of making my

point. Considering the primary lobule, beginning with the respiratory bronchiole, we have -- I warned you this is going to be very diagramatic -- we have some kind of vestibule beyond the respiratory bronchiole connected to that in various air sacs. Now, I won't go beyond that in trying to picture it, but I make this point that down to this point here, concerned with known respiratory air passage ways, the volume of which air got through something like 140 c.c.'s of atypical, and this pass in here adds up to something of that order, and the alveolar spaces to something of that order; now, in the course of inhalation, these are figures representing a relaxed lung. In the course of inhalation, there is expansion down here, and I presume some expansion in this volume also, but even under the most rapid rate of inhalation, the air velocity down in here and out into this space is of the order of, well, at best, one or two feet per minute or one or two centimeters per second.

According to the very elementary view of the thing, from the standpoint of fluid flow, we have to accept the notion that the flow of air into this space is completely slow. There is no turbulence in it, so that as these spaces expand, the air that was in here at the beginning of the inhalation simply recedes quietly out in these spaces here and the new air comes in on top, so that active mechanical

ventilation -- by active mechanical ventilation, I mean active change of gasses, takes place only down into this particular space, since at least under conditions of normal quiet breathing, the tidal volume will not any more than see that and even under heavy breathing of exercise as it goes up, it will still just fall in on top of the receding air.

Now, there is an important point here. The ventilation is all right so far as exchanging is concerned, because the exchange of oxygen and CO_2 across this face, takes place by rapid molecular diffusion, a pretty well accepted notion in respiratory physiology, but the philosophy of diffusion of particles of the order of one or two microns is so slow that we can expect no exchange of particles out into the true alveolar spaces in any way comparable to the exchange of gas out into them, so that these figures that we're talking about here represent deep lung deposition, but not necessarily mean the same for all different sizes, as to the ultimate site.

As particle size goes down, the gravitational defect which is a primary one in causing the sedimenting out of particles, the gravitation effect goes down in size and the diffusion velocity goes up, decreasing size in that fashion, and for simple sedimentation, this is about a quarter of a micron, so it seems to me that basic to our considerations this afternoon, which we have heard so much about

the peculiar behavior of some of these sub-microscopic materials, basic to that may be a deeper understanding than we now have of the actual site of the position and the way that those particles are handled by this total structure, since it seems clear to me at any rate, that a particle that does get out of here by diffusion is going to be - well, say we depend on different mechanisms for its removal, and that is true of particles settled out in the earlier part.

One more point which I'd like to make: If this represents the respiratory cycle with this inhalation and expiration, we know that these early stages of inhalation as a relatively rapid intake which peters off with the end of the inhalation and then there is a rapid beginning of exhalation which tapers off. Current thinking, from recent experimental work, suggests anyway, that the volume of air which is breathed in at this time is represented by this volume of air exhaled, and this volume of air inhaled represented by that volume exhaled and so on, and the so-called sequential ventilation of the first air in, the last air out, which is another way of saying that the different parts of the lungs are expanded at different times in the course of inhalation and exhalation.

Now, the function, among other things, is the time it saves down in the lungs in this volume of air that's being actively exchanged, so that along with this question,

we have also to consider the parts of the lungs that are most actively ventilated during the early phase of inhalation, that they are being last out, as against the air that comes in during the last ventilation and that is the last air out, so that where this air goes to in the lung compared with where this air goes to, is important in your understanding of the site of deposition, and again your point of clearance, so I would like to suggest that more important than our understanding of retention, is a better understanding than we now have of the clearance of the material from the lung and the way in which that is related to the nature, both chemical and physical, of the material.

Now, I had to get my say in. At this point, I should like to open the meeting for discussion from the floor.

BY DOCTOR VORWALD:

Ted, how do you know all this, that is how do you know there is no turbulence of air way out in this microscope?

BY DOCTOR HATCH:

Well, obviously, we can't know it from any experimental work. We can only know it from analysis. We know this, that in the normal typical lung, there are, and we have a pretty good idea, how many, respiratory bronchioles there and we know from anatomical measurements, the cross-section

of air in the anatomical bronchiole, and in one of - one set of data that I have in mind, total air of a whole set of bronchioles, cross-section adds up to one eighth square bronchioles. Respiratory - yes, there are one and a fifth times ten respiratory bronchioles, and that relates to this velocity per second and so on down. This is Landau's chart I think.

These are typical dimensions and reported for the respiratory system. I'm sure they're not exactly right, but they are good enough for our purposes.

Now, one thing we know in fluid mechanics, in the understanding of fluids and flow, and we can draw a sharp distinction between so-called laminal flow and so-called turbulent flow, and determining in advance in any given situation when we're going to have one and when we're going to have the other. The index is the so-called Reynolds number which I won't burden you with, but say this, if a Reynolds number is less than two thousand, you can be sure you have laminal flow.

These calculations here would indicate that in the lungs even under the deepest inhalation at an instantaneous rate of sixty liters a minute, the Reynolds number is tremendous to be compared with two thousand, as a dividing line between laminal and turbulent flow so it's only my faith in the Reynolds number and fluid mechanics that would

tell me that this is so.

I think there is some physiological evidence to indicate that it is so in this case, that after exchange, it's ventilated by diffusion. Does that answer your question?

BY DOCTOR MACHLE:

Mr. Chairman, I'd like to compliment Doctor Hardy on a very excellent summary of the beryllium problem. I would like also to ask if she would elaborate on the cases presumably exposed to low beryllium phosphor. We have been following data on several thousand people exposed to low beryllium phosphors since 1945. From our experience, from the data we have now pooled together, we have yet to find any cases of exposure to beryllium encountered in the low phosphor, at least in one manufacturing operation in this country, and in another manufacturing operation, there have been several hundred including - several hundred thousand man hours of exposure to low beryllium phosphors, the percentage was less than three years.

BY DOCTOR VORWALD:

The percentage was less than what?

BY DOCTOR MACHLE:

Beryllium.

BY DOCTOR HARDY:

My data is a mixture of material that I have by

the courtesy of Doctor Kline, on General Electric phosphor which, I understand, is always below four percent, and he reports about eleven cases, and we have two cases in Massachusetts in or from subsidiary companies in that same phosphor. (NOTE: Doctor Hardy requested following the closing of the session that the reference to General Electric be deleted from her remarks. However, as the reporter understood the response, the reference is made in such a manner that it is felt Doctor Hardy should make the deletion in such a way as not to confuse her statement).

I have reports from Nash in England, reporting six cases of chronic beryllium poisoning, three of these from low beryllium phosphor manufacturing. I used the term 'roughly' advisedly, because I didn't have the precise data as to the total number of these, but it was something of the order of fifteen which is on the low side, of individuals with an unknown number, doubtless in the thousands, exposed to phosphor below four percent. Is that what you want?

BY DOCTOR MACHLE:

May I ask again - yes, that's a very significant observation, but there are several various significances in beryllium phosphor quite apart from what Doctor Pratt mentioned, characteristic, so to speak, of exposure. By the characterization of phosphor, the total amount of beryllium

present is an important consideration because of the limitation in the limit of the solution which you can make of beryllium in material.

The second variable is the completeness of the reaction which, of course, is determined by the time, temperature and repetition in firing, together with the use or non-use of flexes. In situations where we have encountered cases which seemingly occurred from exposure to low beryllium phosphor only, it has been possible in at least two of the situations to establish that there have been unusual practices in the preparation of phosphor by - in the direction of lessening firing, experimentation with flexing methods, and so forth, which lead one to believe that it was an anomalous situation and it could not be included in the general epidemiological consideration which, obviously, you are bound by, to have a certainty as to the nature of the exposure.

BY DOCTOR HARDY:

That is why I quoted you, Doctor Machle, as one of the more studied of physical characteristics of state of *aggravation*, and so forth, and so forth, but it seemed to me in reviewing the evidence in my own personal experience, we had to say that the low beryllium phosphor used in that manufacture before May 1949 had caused the death of a number of cases of beryllium poisoning and I think, as you do,

this is a very crucial point in studying the epidemiology of the disease.

BY DOCTOR VORWALD:

Mr. Chairman, it may be interesting for you to know, I think we have in recent years, or last year, succeeded in reproducing a lesion in the lung of animals which simulates, at least, the lesion which we see in the human cases exposed to beryllium, and certainly the simulation is as comparable in that instance as is the simulation between silicotic produced and the silicotic natural subjects. We have succeeded in doing that after many months of exposure to pure beryllium oxide at extremely low levels of concentration, and also to a beryllium sulphate at extremely low levels of concentration.

There is - in my own mind, however, there is one difficulty which I can not explain and that is that we have not as yet succeeded in reproducing the number of lesions that we see in human cases. Now, I must admit that the lesions that we have reproduced experimentally are very few and far between, but we will be reporting some of our other observations on Wednesday when we discuss the pulmonary cancer and its relationship to some of our exposures in experimental animals.

I should like to ask, if I may, just one more moment, Mr. Chairman. Doctor Solandt - we may have not given

him sufficient time, and if he could merely tell us very briefly, what he is doing then as the agent that is producing this change in the group - in these experimental animals, and also related to human subjects in the abrasive bauxite industry. Is Doctor Solandt still here? (No response). Doctor Shaver, could you - he's not here. We may have stopped him too short; I'm not quite clear as to his final conclusion. Perhaps while Doctor Shaver is asking for that, some others might have a question.

BY DOCTOR GREENBURG:

Mr. Vorwald, I'd like to ask Mr. Hatch a question. In that diagram, you've got there for the inhalation and the exhalation, you say the material can be fractionated to different parts of the cycle, but as I remember it, you said that the total retention was about one tenth of a percent up to one percent.

BY DOCTOR HATCH:

The ultimate retention.

BY DOCTOR GREENBURG:

Yes, the ultimate retention. Now, if the ultimate retention is one tenth to one percent and the concentration to which the animal is exposed is approximately uniform, for any given duration of time, wouldn't the amount retained in the various parts of the cycle be approximately the same in the last analysis?

BY DOCTOR HATCH:

Well, I think somebody who knows more about the lungs than I do would have to answer that. The picture I have is that there are - there is relative ease in ventilation of certain parts of the lungs compared with others and you would expect, therefore, over a period of time to find a disease and, therefore, there would be a higher deposition in such areas.

BY DOCTOR GREENBURG:

No, that isn't the thing I was talking about; the first thing I'm quite in agreement with.

BY DOCTOR HATCH:

You're speaking about this chart on the right?

BY DOCTOR GREENBURG:

That's right.

BY DOCTOR HATCH:

This is inhalation-exhalation-ventilation curve for the lungs.

BY DOCTOR GREENBURG:

Right. Now, it seems to me that the retention in all parts of that cycle, in all parts of the inhalation cycle would be approximately the same.

BY DOCTOR HATCH:

Well, I was suggesting -- no, I can't say that is so. I was just suggesting it. You say this, the subject

breathing fifteen respirations a minute, then this is four seconds from here to here. The average duration of stay of this air in the lungs will approach four seconds. The average duration of stay of this air in the lungs will be one second or less. Now, the amount that's deposited out of that air is going to be in proportion to the duration of stay in this actively ventilated space so that in certain portions of the lung, favorably receiving this air, then they're going to get more dust.

Dr. Kehoe →

UNIDENTIFIED SPEAKER:

Gentlemen, I suggest that you're talking about two different things; he is talking about retention in respiration and you're talking about ultimate retention in the tissues after the elimination process.

BY DOCTOR HATCH:

Maybe I'm a little confused.

Dr. Kehoe

UNIDENTIFIED SPEAKER:

You can get particles of this order, magnitude, something of the order or magnitude of two to three microns; you can get upwards of thirty-five or more percentage retention in the lung during the respiratory cycle. How much you'll find there six months later is a totally different story.

BY DOCTOR HATCH:

But with respect to that, that average figure is

made up of higher percentages in the lungs and lower percentages in others depending on the activity of ventilation.

BY DOCTOR VORWALD:

Ted, are you sure that the first air in is the last air out?

BY DOCTOR HATCH:

I think the most - the best evidence we have on that is a paper by Fowler, remember, down in Pennsylvania, in which he determined that was so by the ingenious trick of splitting up the inhalation for a moment and then suddenly completing the exhalation with a mixture, and then he analyzed the exhaled air in the same way and then he found that that same mixture going in was found in the air coming out. Now, we have some other indirect evidence and we have a little bit of evidence that agrees with Fowler.

BY DOCTOR VORWALD:

Doctor Shaver?

BY DOCTOR SHAVER:

Rather than hash what Doctor Solandt had to say, I will just read part of his summary and conclusions.

(Doctor Shaver reads from Doctor Solandt's paper, which has been filed with the Saranac Laboratory.)

UNIDENTIFIED SPEAKER:

I wanted to ask Doctor Hardy a couple of questions. One is what is the longest period of time for treatment of

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Canada

C. G. SHAVER, M.B.
SUPERINTENDENT

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SECRETARY-TREASURER

MISS MADGE McCORT
SUPT. OF NURSES

P. O. BOX 158



ST. CATHARINES 17th November, 1953.
ONTARIO

Doctor Arthur J. Vorwald,
163 Park Avenue
Saranac Lake, N.Y.

Dear Doctor Vorwald,

I have had the paper which I presented at Saranac Lake a year ago, re-typed, and the pathological cuts are included. There has been no change in the text.

I hope that any delay on my part has not inconvenienced you.

Yours sincerely,

C. G. Shaver, M.B.
Superintendent.

CGS:LCP.

SEVENTH SARANAC SYMPOSIUM

BAUXITE FUME PNEUMOCONIOSIS

by

C. G. Shaver, M.B.

September 1952.

BAUXITE FUME PNEUMOCONIOSIS

by

C. G. Shaver, M.B.

On the occasion of the Sixth Saranac Symposium, Dr. A. R. Riddell presented a very concise, but clear-cut picture of the clinical features of Bauxite Fume Pneumoconiosis. Chemical aspects of the disease were discussed by Doctor C. M. Jephcott. I have been asked to discuss the clinical developments since that time.

Dr. Riddell reported 30 cases of well-established disease, 29 early and 17 doubtful. The present situation reveals that we have to date a total of 38 cases of well-established disease, some of these were previously classified as Early. In the Early group there are 65 cases, and there is a further group of 23 Doubtful. It is quite impossible, because of changing conditions of employment, and lay-offs to formulate any opinion as to the incidence of disease among those employed. One of the well-established cases, who had not been x-rayed previously, reported recently at a mass survey. He had been an aluminum oxide furnace feeder from September 1939 to March 1942, since when he has worked as a farmer. He has no complaints, and his examination, other than the x-ray of his chest, was negative. A case diagnosed as Early disease in 1951, had had two years exposure as a hot change man in 1942 and 1943, but had missed being x-rayed since then, although his employment since 1943 was not associated with furnace fumes.

Deaths to date, which have been ascribed to the disease number 13, or 34.5% of the group recorded as Well Established. Although spontaneous pneumothorax has been a very

common development in the group, it has been responsible for sudden death in only one case. This patient was recovering from a unilateral pneumothorax when he developed a massive pneumothorax on the contralateral side, and died within a few minutes. In two cases infection played an important part as a cause of death, one showing a rather marked downhill course following pneumonia, and the other developed an infection which terminated in gross bronchiectasis. The remainder died of ^{chronic} cor pulmonale.

Eight deaths have occurred in the group from causes unrelated directly to the pulmonary disease, two of these from coronary occlusion, one from brain tumour, and one from purulent pericarditis. We were not successful in obtaining post-mortem examinations in the remainder (T.M., F.P., W.M., and R.M.).

Special investigation was carried out on one case, who in 1947, during the development of his disease showed a rather marked paradoxical movement of his mediastinum (A.M.). Later however, the intensity of emphysema in his upper right lung area seemed to compensate, and this phenomenon became less marked. He was admitted to the Toronto General Hospital in March 1950 for investigation of cardiovascular effects which may have been initiated by his pulmonary fibrosis. At this time he had a normal cardiograph. After catheterization of the heart it was concluded that there was a slight increase in the degree of negative pressure within his thoracic cage, and a slight degree of pulmonary hypertension.

Interestingly enough this man was totally disabled in 1947, but improved to the point that he wanted to do something, and he accepted our advice to take a business course, which he completed. He then obtained a light job as timekeeper at a local plant, and from 1950 to December 1951, he was quite well. He then developed an acute pulmonary infection, from which he has not completely recovered. The clinical improvement between 1947 and 1950 corresponded with increasing emphysema in the right lung, and I feel that had the cardiovascular investigation been carried out in 1947, the changes noted would have been more marked than in 1950.

Detailed lung function tests have been carried out on two patients (A.M., R.S.,), one being the case reported above. He was unable to complete the lightest test given on the ergometer, as well as being unable to do the other exercise test of stepping up and down on a low stool. His disability in 1949 was rated at a minimum of 75%. As stated above, however, the man's tolerance for work seemed to improve later.

The other case (L.R.), on whom lung function tests were carried out in May 1948 required an alveolar oxygen concentration of 59.2% to give 100% artereal oxygen saturation. The time required to reach 100% saturation on changing from room air to 100% was 77 seconds. This compares to a series of normal subjects who had given a mean O₂ concentration of 30.1% for 100% saturation, and a saturation time of 43 seconds. His actual vital capacity was 1.1 litres, and calculated normal V.C. 3.9 litres. His Electrocardiograph at this time showed no evidence of right heart failure. This patient is still alive, although he lives a most sedentary sort of existence.

CORTISONE

It was with some anticipation following upon the results of the use of Cortisone in Beryllium disease, that the Workmen's Compensation Board permitted us to use the drug on selected cases of Bauxite Fume Pneumoconiosis. The case recorded above was treated from January 11th 1951 to February 12th, 1951, with 150 mgm. of Cortisone, for 7 days, and 200 mgm for the remainder of the period. During the time of treatment, there was no change in his subjective symptoms, if anything his dyspnoea increased slightly. This was ascribed to a gain of 10 lbs. in weight while he was taking the drug. Since discontinuance of the therapy his weight has returned to its former normal, and no change has been noted subjectively or objectively in the patient. We have not used the drug on other patients.

PATHOLOGY:

A previous report on the pathology of Bauxite fume Pneumoconiosis has been made by Wyatt and Riddell. We have had the opportunity since this report to study two cases in which death occurred in patients showing early lung changes. The first died on January 2nd, 1949. He had a stone in his common duct and vegetative endocarditis. The pathologist could not state the exact cause of death, but was quite certain that the pulmonary condition was in no way responsible. The second case died of coronary thrombosis in December 1951.

Case 1.
(W.M.)

age 40. He gave an occupational history of 19 years in one of the local abrasive plants. From July 1927 to January 1928 he was in the mix room, after which from January 1928 to March 1941, he was an

aluminum oxide furnace feeder, with the exception of a two-year lay-off. Following this he was a supervisor in the furnace room.

The first x-ray taken on this man was in September 1944. It was reported that there was a light increase in the first and second interspace markings, but the change was not sufficiently marked to make a diagnosis of occupational disease. In April 1947 these lung changes seemed to be of sufficient importance to make a diagnosis of early disease.

The last films taken on him were in July 1948 and these show light granular infiltration above the third rib on either side. Although of better technique, they show some increase in the lung shadowing over his initial film of September 1944.

GROSS PATHOLOGY:

Dr. J. P. Wyatt, Pathologist at the Toronto East General Hospital reported that the pleural surface was smooth. The lungs were bluish grey in colour, with fine dark-blue mottling. On section, the lung parenchyma was pale-grey in colour. It had a fine spongy appearance. No abnormality was noted in the bronchi or vessels.

In the right upper lobe, near the apex, there was a small area which felt firmer than the surrounding tissue. Fine interlacing strands of dark grey tissue could be seen, and between these slightly dilated air spaces. This was the only area in the right lung which appeared abnormal.

On the left in the upper lobe at the apex, was a small area where the alveoli were dilated, giving the appearance of a honeycomb. Adjacent to this area were seen small patches which were dark gray in colour, and firm to touch. The remainder of the lung had a normal appearance.

Microscopic Description:

The lung parenchyma showed a diffuse uniform interstitial fibrosis throughout. The normal alveolar septal lace work is replaced by a stiffened collagen deposit, some of this is hyalinized giving a filigreed trabecular pattern. The septal thickening of the right lower lobe is minimal, but the stiffened radiating septa are distinct. The other lobes are more or less in the same stage with a moderate degree of septal thickening. At places the septal fibrosis passes into irregular masses of scars, radiating from which are bands of hyalinized septal tissue. A large area of scarring with trapped anthracotic dust is noted in the right upper lobe which as described grossly, is firmer in consistency than the surrounding tissue. Nowhere is fibrous nodulation found, nor is there any evidence of koniophthisis. Emphysema is not marked, though some dilated spaces are found along with fibrosis. Inflammatory reaction and oedema are patchy. The cellular reaction is predominantly small round cells, and in some areas polymorphonuclear leucocytes, which are more intravascular than interstitial. The small blood vessels have also a thickened hyalinized wall.

The lymph nodes show fibrotic scarring and anthracotic pigment deposition. One lymph node shows three minute foci of a cellular demarcated amorphous debris, reminiscent of healed tubercle.

Opinion:

1. The upper lobes are not the only lobes involved.
2. The trabecular outline is that of single stiffened septal walls.

3. Curiously enough the collagen is all of the same age.
4. Previous lungs which have been sectioned have shown a more heterogenous morphology.
5. The lesion of increased septal cellularity is not present.

Analysis of lung ash:

Silica: 28.95%

Alumina: 16.86%

0----oOo-----0

Case 2.

R.M.

age 57. This man gave an occupational history of working as a general labourer in the arc-furnace room of one of the Plants from 1935 to 1940. He was then a crane operator in the same department until the time of his death.

The first chest film taken on him was on May 2nd, 1945. It shows some suggestion of increased markings in both apices, although the change was not of a degree as to substantiate a diagnosis of occupational disease.

Stereoscopic films were taken in April 1947, and these showed a very light, but quite distinct granular type of infiltration above the second rib on either side, and some slight increase in his lung markings in general.

Further films did not reveal any definite extension of his disease. The last plate on file is dated September 1951.

The films in this case are not shown, because it is felt that the disease would not show up to advantage in a small print.

The pathological report on this case is presented through the kindness of Doctor A. J. Blanchard, Sunnybrook Hospital, Toronto, Ont.

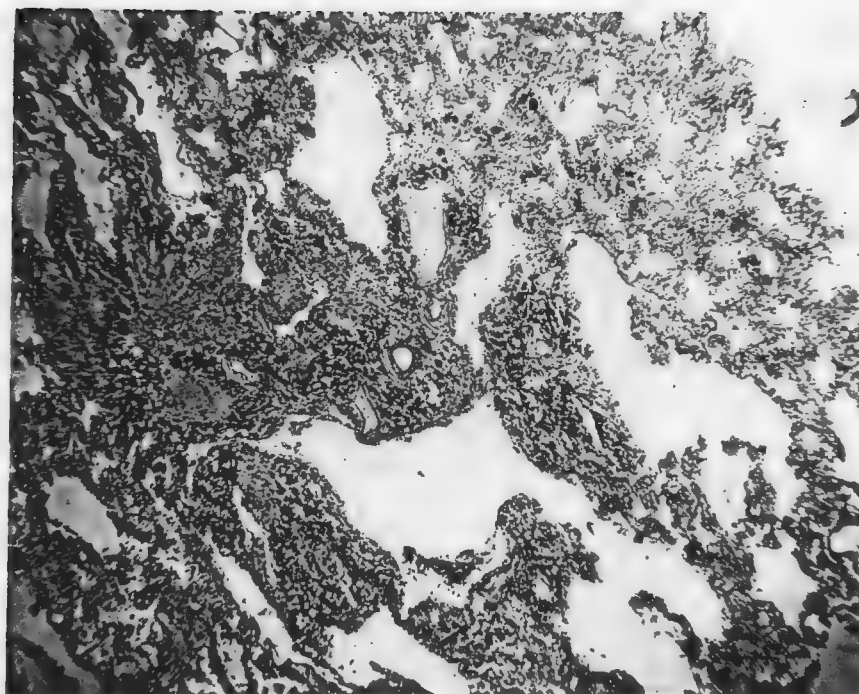
Gross pathology of the lungs showed that the pleura was generally smooth, glistening and greyish in colour, with black mottling. The lungs were slightly emphysematous, but no bullae were noted.

Cross section through the lungs showed a fine moderate diffuse dilatation of alveoli, so that the lungs were more porous than normal. In addition there was considerable black patchy pigmentation throughout all lobes. These pigmented areas however, were not indurated. Occasional areas showed small rounded nodules, one to two mms. in diameter, which were not considered typical of silicosis. Occasional nodules in a perivascular or subpleural locations were larger and rubbery in consistency. At the extreme right apex, there was a superficial firm cartilagenous like nodule, .5 cm. in diameter. On the left there was a suggestion of a diffuse, interstitial fibrosing process involving the central portion of the upper lobe. Peribronchial lymph nodes were small and blackish in colour, and soft in consistency.

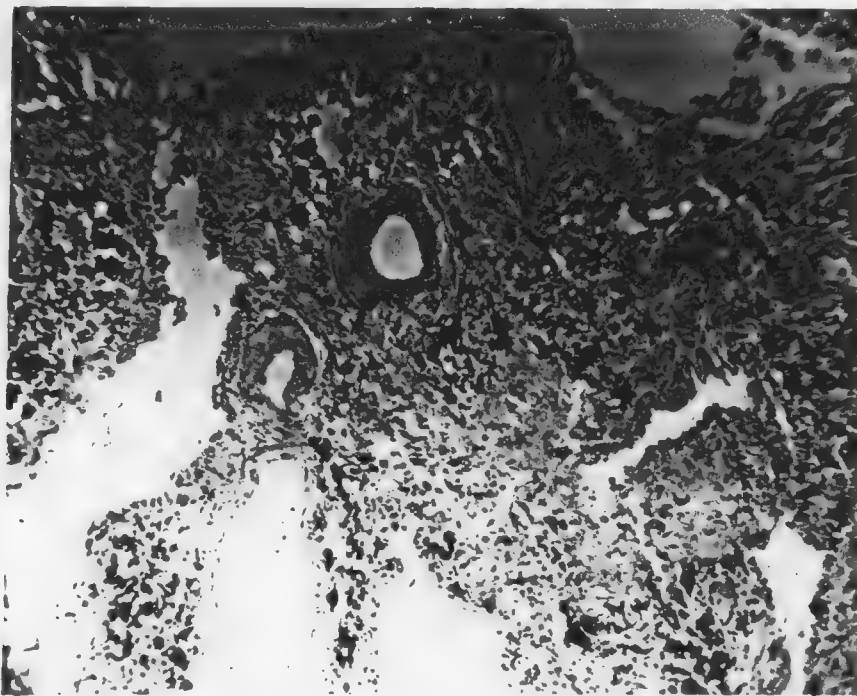
Microscopic examination:

Right lung

(a). This shows patchy areas of scattered dense dark anthracotic pigmentation with a slight amount of underlying dense fibrous tissue. This however, is not showing any circumferential arrangement, such as is seen in silicotic nodules. In addition almost all of the alveolar walls are slightly to moderately thickened due to an increase in collagenous fibrous tissue. The alveolar spaces show a patchy dilatation, and many of them contain histiocytes some of which have engulfed anthracotic pigment.



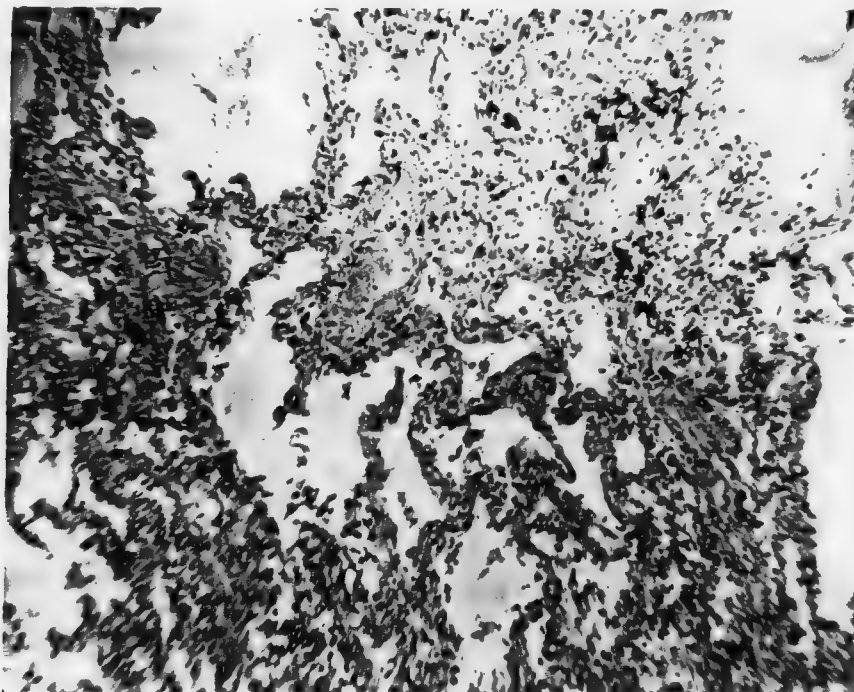
A.49 Low power view of lung showing patchy fibrous thickening of alveolar walls and entrapped anthracotic pigment. In one area the collagen is hyalinized, but there is no evidence of any nodulation. There is moderate dilatation of some of the alveoli.



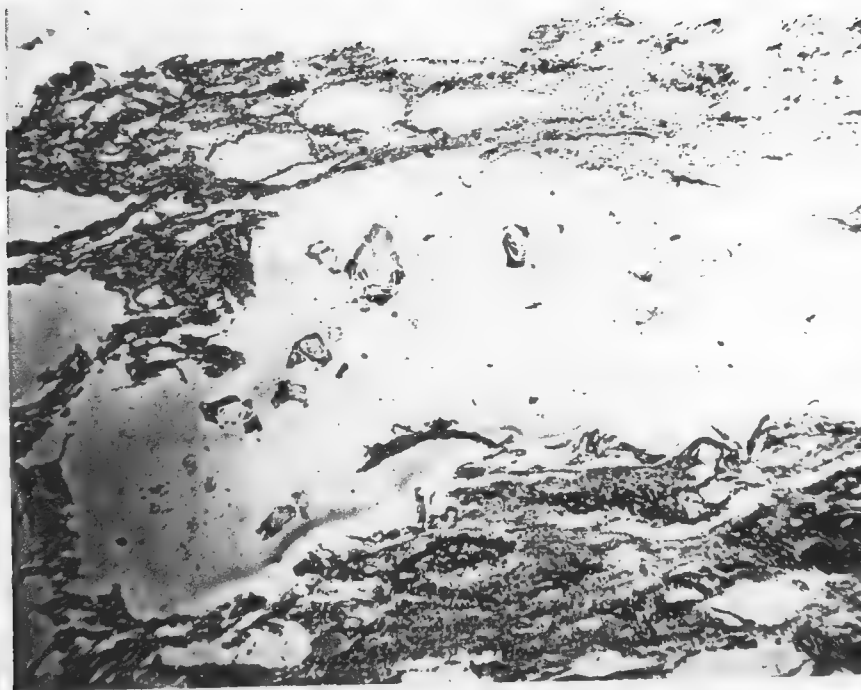
A.48 High power view of small area of A. 49 showing again the interstitial, non-nodular fibrosis, pigmentation, and some thickening of small arteries.

(b) Two further sections from the right lung showed the pleural surface of the lung, with a thickening of an interlobular septum by dense fibrous tissue, and also patchy subpleural and perivascular anthracotic pigmentation, and a thickening of the alveolar walls with emphysema and patchy areas of atelectasis. One of the sections also includes a fairly large bronchus which appears to be essentially normal apart from a very slight chronic inflammatory cell infiltration, and shows a peribronchial lymph node with considerable anthracotic pigmentation, and some fibrosis, which however, was more diffuse, and not suggestive of the silicotic type.

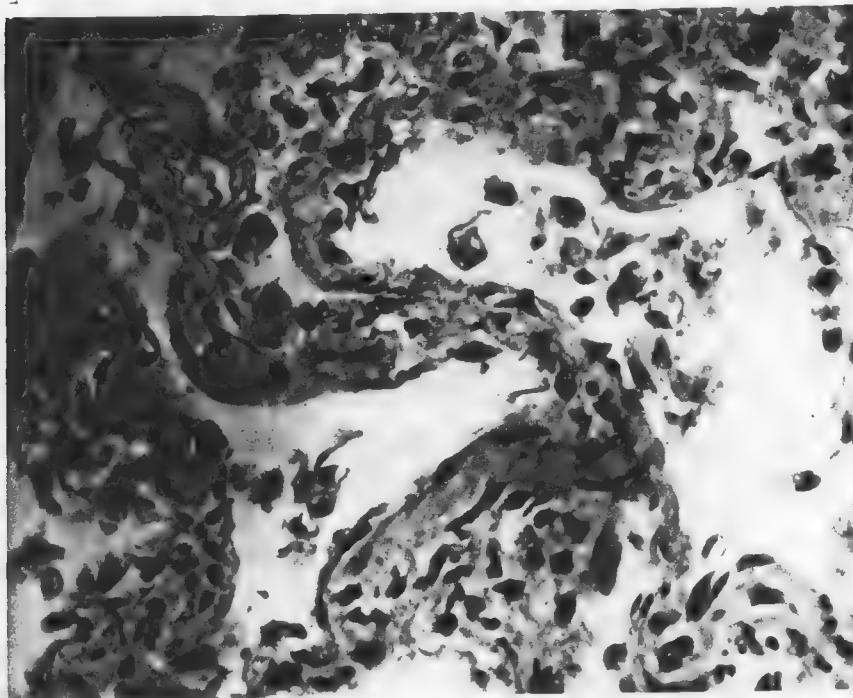
(c). Two sections from the left upper lobe show a marked degree of interstitial thickening of alveolar walls. Many of the alveoli are collapsed, and in addition, most of the persisting alveolar spaces are filled up with an accumulation of histiocytes. Here and there, were dense accumulations of black anthracotic pigment.



A.50 This is a low power view which shows a more diffuse fibrous thickening of alveolar walls, and in some of the alveoli there are collections of phagocytes



A.51. This shows an emphysematous bulla. The surrounding lung is partly compressed. You can also perceive patchy increase in fibrous tissue in the alveolar walls in the interstitial tissue.



A .52. You can see the increase in fibrous tissue, both fibroblasts and collagen being laid down in the alveolar walls in increased amount.

Diagnosis:

- 1). Diffuse interstitial fibrosis of lungs, moderate.
- 2). Emphysema of lungs, moderate.
- 3). Anthracosis of lungs and peribronchial lymph nodes, moderate.

Summary: Examination of this tissue disclosed that the obvious cause of death was a recent coronary thrombosis with myocardial infarction. The lungs showed an unusual picture in that there was a very diffuse interstitial fibrosis with thickening of almost all of the alveolar walls and patchy atelectasis and emphysema. In the atelectatic areas, many of the alveolar spaces were filled with dense accumulations of histiocytes. Nowhere was there any suggestion of the lesions of silicosis. The pathological changes described above are compatible with those seen in early cases of Bauxite pneumoconiosis.

Analysis of Lung Ash:

Silica: 22.3%

Alumina: 32.41%.

These cases differ from those described by Wyatt and Riddell in as much as they did not have the thickened pleura. Gross bullous emphysema was lacking, although both cases show beginning changes of this nature. Interstitial fibrosis, although present, is much less marked than in the cases described by Wyatt and Riddell.

TUBERCULOSIS:

A total of 11 cases of tuberculosis from the plants under surveillance, has been treated. In only one of these however, was there x-ray evidence of pneumoconiosis associated with the tuberculous condition. This has been treated, and the tuberculous process is progressing favourably. He is disabled however, because of his pneumoconiosis.

In the remaining group, all but one have been discharged from hospital, this patient having been diagnosed only within the past few months.

It would seem that there is no evidence to suggest that tuberculosis has been more frequently encountered in this Industry than in other Industries of the district. The presence of Bauxite Fume Pneumoconiosis made the treatment of the Tuberculous condition in the patient where both were present, somewhat more difficult, but the ultimate progress of the tuberculous disease has been satisfactory.

PREVENTION:

The Industries concerned, in co-operation with the Department of Industrial Hygiene of the Province of Ontario have effected a marked improvement in the ventilation of all Plants. This unquestionably has been of major importance in decreasing the incidence of the disease. In addition to this, periodic physical and x-ray examination of employees in the Industry has been carried out; at first at intervals of six months, latterly, except in special instances, it has been done once a year. There has also been improvement in the method of feeding furnaces, so that the men get less exposure to furnace fumes.

A very rigid pre-employment examination has been enforced, and men who have had previous exposure as hard-rock miners, as well as those who show chronic pulmonary disease, such as bronchitis or bronchiectasis have been excluded from employment.

The disease Bauxite Fume Pneumoconiosis was first identified in 1942, and it would appear that the major occurrence of the disease among workmen associated with Bauxite Fumes was between the years 1942 and 1945. Since then there has been a gradual lessening of the incidence of disease, and it is felt latterly that the disease is under control, although there still remains a goodly number of workmen, who because of previous exposure, show distinct lung changes.

these patients you have referred to?

BY DOCTOR HARDY:

One recently reported by Kennedy in the Journal of the American Medical Association, I have continued to treat that same case up until now.

UNIDENTIFIED SPEAKER:

The second question, one of your patients showed here, looks like a hyperthyroid; is there any indication toxicity stimulates the hyperthyroid, or is it because of hyperacidity of the bone?

BY DOCTOR HARDY:

We don't know; there has been a lot of speculation about that. We had two cases of thyroid tumors that sort of disappeared. They had high m. p. r.'s. No one every explored their lungs to know. Hypercalcemias were present. Our feeling is that, in some mysterious way, the behavior of beryllium in the body, particularly its disposition and modus operandi must be related, but at present I have no knowledge except those early tracer studies of Doctor Hamilton Berkeley, except that when he gave beryllium, the material went in the animals' body and was in the liver and kidney for a while but it never left the bones for the duration of his study. That's been true in autopsy cases, where beryllium was present. Doctor Fuller Albright thinks that the renal calculi and perhaps the other phenomena that I have

enumerated are related, just as he thinks they are in sarcoidosis to a protean binding, that is protean will bind calcium in certain diseases, but as far as I'm concerned, that's a description, not an explanation. I don't know.

UNIDENTIFIED SPEAKER:

What is the dosage of your ACTG for that treatment?

BY DOCTOR HARDY:

Well, it varies a great deal. In general, we have given him 200 milligrams a day, ACTH embodied into fifty milligrams every six hours, for a few days; then down to a hundred milligrams spread out, then shift to the oral cortisone, keep him there one or two weeks until you're sure that you have what would be the best possible benefit from the symptomatic standpoint, and in various cases, we have tested this by pulmonary function study and cardiac catheterization study and then we do what Doctor George Dorn calls titrate the dose, meaning let him go down to the point below which he is still comfortable and then keep him there.

I'm a great deal older than I was when I talked about this in 1950 and I have patients that have been on 75 milligrams of cortisone, 25 milligrams every eight hours for a year, and I can't see where I've done any harm, and I've taken symptomatic patients from a complete chair patient back to forty hours a day - four hours a day, and this is very impressive even though in all honesty, I don't

think I'm curing them.

UNIDENTIFIED SPEAKER:

The only reason I'm asking, Jerry Kane of Michigan seems to think that the use of cortisone may lead to sporadic sclerosis; I don't know.

BY DOCTOR HARDY:

Well, I think it might easily be true, but I think in the case of beryllium poisoning, you have to sort of take the choice and play your cards as you go.

BY DOCTOR VORWALD:

Is that a general tendency in cortisone?

BY DOCTOR HARDY:

Yes, it's been my experience in talking with Doctor Max Michel, who has treated very many of these, that you may get brilliant results in treatment of sarcoidosis with ACTH or cortisone, but as I pointed out in that swift-running survey summary, just given you, I have seen cases of sarcoidosis that simply will not respond in any way to ACTH and cortisone, and I have seen - I have even seen cases recently -- two are very fresh in my mind -- that have definitely been made worse, and have proven this by pulmonary function studies and a clinical downhill course of obviously increasing replacement of the granular tissue. This I have not seen in beryllium poisoning except for my remarks that one or two cases I have watched appear to be getting, from the

laboratory standpoint, worse though they're systematically better.

BY DOCTOR TABRUCK:

I'd like to state that our experience in the treatment of chronic beryllium poisoning with cortisone parallels that of Doctor Hardy. We have seen several dose cases under cortisone therapy and we find we have been able to do a great deal for these patients, but we find very definitely we're not curing anything. We find the patients go downhill and the best we can offer them is to give them a more protracted period of disability but a more practical period of disability if you can use that word. In other words, they are sleeping much more comfortably during this period of disability, which becomes longer, but the end results are about the same.

I'd like to ask Doctor Hardy what, in her opinion is the activating factor in her precipitate mechanism? Is it a matter of true allergy or is it a matter of physiochemical changes within the system that precipitates the instance of disease in beryllium poisoning?

BY DOCTOR HARDY:

Well, I see I didn't make myself clear or talked too fast. I think the beryllium is in the body very often in happy equilibrium, stored for excretion, and then something happens to the patient -- maybe it's a respiratory

tradit infection or this very fascinating business of the woman having a baby and getting along quite well, and then when the baby is four or five months, she begins to go downhill in induced weight loss. I have seen this very strikingly, or in unusual exercise, the soldier, you remember a very small but definite group, after experience in combat; or I have young girls in cases where, or cases where a lazy 190 pound man went and was put through boot training and lost thirty pounds and he became a clinically active case of beryllium disease.

In fact, in practically all of the cases in which I have been able to make a good clinical hunt, I have been able to find something that happened to that man or woman after he or she stopped inhaling beryllium or perhaps still is in the situation that is an added insult or added physiological burden, that if you like, makes it possible for the beryllium to become pathogenic.

I suspect you're referring to the very fascinating work of the boys, showing that beryllium in the test tube and in small animals will inhibit magnesia, will take the place of magnesia in certain enzyme systems and, by pressing out the phosphatase, set up a whole chain reaction so to speak, and it's very, very seductive to consider that this may be the means by which to say your response takes place, that later on you'll have fibrosis and so on, symptom

producing. The cortisone that is used in test tubes on small animals compared to the human of this particular set-up, makes it a little difficult to translate it from the test tube to humans, but there it is possible for later use.

I haven't seen any evidence there that the clinically active disease is an allergic type of response, although I think Doctor Sterner has a very good basic broad idea that may explain a lot of things about goiter response to toxic insult, not just beryllium, but also infection.

BY DOCTOR HATCH:

I think at this hour I'd like to call the meeting to a close, and I'd like to make one comment in closing. We have heard, this afternoon, of the action of different dusts in the lungs, the relative importance of different factors in the dust, that go to determine the nature and magnitude of the action. Very illuminating, I believe, in the light of this morning's discussion in which we had great difficulty in arriving at definitions to a high degree because there were so many gaps in our knowledge. I should like to leave with you this thought, will the day ever come when we will be able to anticipate difficulties associated with the inhalation of particulate matter and not have to wait until the difficulty has developed before becoming concerned with it. It's going to the question or the degree of which we can reduce our understanding to the fundamentalities, the common

denominators that go to make up the problem, whether it's the problem of asbestos or bauxite or whatever, to whatever degree we can meet those fundamentals and extract out of them a common denominator.

Is it going to be possible for us to anticipate and predict, from basic studies of new dusts, the possibility of toxic infection? I, for one, would be pretty discouraged if I thought that day would never come. I don't want to suggest that one day we'll be able to explain everything, but I certainly hope we will be able to do a better job than we have in the past in anticipating this trouble.

The other comment I want to make is one I think Doctor Fletcher spoke in particular about this morning, that in our understanding of these problems we have to depend as much on epidemiological findings from the field as we do on the findings from the laboratory. One complements and supplements the other.

I think, with that, we'll call the afternoon meeting to a close.

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(Session adjourned at 5:45 P. M.).

REPORTER'S NOTE: Throughout the transcript, unless names were specifically spelled at the time given, all proper names will be subject to change or correction.

✓ Edited page 77

DR. FRIEDMAN: Roughly from 25 to 86 years of age.

Edited page 90

Wright ✓
DR. FRIEDMAN: Dr. Wright, how many patients in the dusty trade groups you studied had radiologic evidence of pulmonary disease and how many were disabled according to present standards of pulmonary function studies?

✓ Edited page 90

✓ DR. FRIEDMAN: You may have been dealing with places where the improvement of dust conditions had already been accomplished. Do you know how many years these men worked in hazardous concentrations of dust and how many years they did not?

Edited page 112

DR. FRIEDMAN: I do not believe as so many have inferred that the administration of justice in cases involving occupational diseases of the chest is

hampered by dishonest doctors and lawyers. I believe that 99.44% of them are basically honest people. The major difficulty we doctors and lawyers encounter in attempting to solve intelligently and equitably claims for compensation is the fact that we are forced to render opinions on the basis of our current knowledge in the face of antiquated statutes which were enacted 5, 10, 15 or more years previously. We make considerable progress in medicine each year and I think that much of the difficulty could be resolved if the compensation acts of the various states were sufficiently flexible in nature to permit the application of newer and acceptable medical knowledge from year to year. Yesterday, for example, Mr. Waters referred to the West Virginia Compensation Act which he believed was a very good act; but in West Virginia, if my information is correct, occupational diseases of the chest are limited to those cases in which nodules can be identified in the roentgenogram. Today we know that there can be very significant and disabling pulmonary dust disease

without any evidence of nodulation in a roentgenogram of the chest. I can see how in West Virginia a doctor for the claimant or a doctor for the defendant would have great difficulty in resolving their problem in the case of an individual who is disabled from pulmonary dust disease and so proved to be disabled but who has no nodulation in his chest roentgenogram. Finally, what kind of disability are we talking about - economic or medical?

October 28, 1952

Miss S. Mulstein
Secretary to -
Mr. M. Eisenbud
United States Atomic Energy Commission
P.O. Box 30, Ansonia Station
New York, 23, New York

Dear Miss Mulstein:

Thank you for your letter of October 18 in which you enclosed a copy of the talk given by Mr. Mulstein at our Seventh Saranac Symposium held here in Saranac Lake last month. We are very grateful to you for sending the paper to us so promptly.

Sincerely yours,

Arthur J. Vorwald, M.D.
Director, The Trudeau Foundation
and The Saranac Laboratory

AJV:gn

UNITED STATES
ATOMIC ENERGY COMMISSION
NEW YORK OPERATIONS OFFICE

ADDRESS REPLY TO:

MANAGER OF OPERATIONS
U. S. ATOMIC ENERGY COMMISSION
P. O. BOX 30, ANSONIA STATION
NEW YORK 23, NEW YORK

AND REFER TO:

HS:srm

October 15, 1952

Dr. Arthur J. Vorwald
Director of Research
Saranac Laboratory and The Trudeau Foundation
7 Church Street
Saranac Lake, New York

Dear Dr. Vorwald:

As you probably know, Mr. Eisenbud is at present abroad for a period of about 8 weeks, and therefore, I am taking the liberty of forwarding to you a copy of the talk he gave at Saranac recently which he indicated you would need for your records.

Sincerely,



S. Mulstein
Secy. to Mr. M. Eisenbud

Enclosure:

"The Fate of Inhaled Particulates"

VORWALD COLL
BOX 89-91

SEVENTH SARANAC SYMPOSIUM

THE SARANAC LABORATORY of the
Edward L. Trudeau Foundation,
Saranac Lake, New York

September 23, 1952
Session Commencing 9:00 A. M.

PNEUMOCONIOSIS IN COAL MINERS

Chairman: PHILIP DRINKER, Sc. D.

Industrial Hygiene Studies of Coal Miners in
Two Geographical Areas in the United States

E. D. J. Urban

Discussion

Radiological Classification

Charles M. Fletcher, M. D.

Discussion

Pathology of Coal Workers' Pneumoconiosis

Arthur J. Vorwald, M. D.

Discussion

Epidemiology of Coal Workers' Pneumoconiosis
in Wales

Charles M. Fletcher, M. D.

Discussion

BY DOCTOR VORWALD:

It's past our time to begin and there are a few announcements which I should like to make before the formal opening of the morning session.

Again, to remind you, those of you who have not registered, please do so at the John Black Room. Also, tomorrow night, on Wednesday, at eight o'clock, there will be a demonstration of colored photography by the Ansco Company in the main ballroom of the Saranac Lake Hotel. Those of you who are interested in color photography, I know, will be interested in what they have to present. Also,

there are a number of you who are Rotarians and today being Tuesday, the Rotarians will meet this evening at the Fish and Game Club at six o'clock. The Fish and Game Club is on the outskirts of Saranac Lake and should you inquire, I'm certain that you will find your way there. It sounds very interesting when the Rotarians meet at the Fish and Game Club instead of at noon time at the hotel, because it always means something extraordinary in food and other things, so if you wish, you're welcome to go there, to make up your Rotary.

The banquet on Thursday night, as you will note from your agenda, there is to be a banquet on Thursday night, and I wish to announce that ladies are cordially invited to the banquet. There will be a good many there, so those of you who are accompanied by a lady, please bring them.

With that then, we will enter our - or begin our sessions for this morning, and our Chairman certainly needs no introduction to you -- Professor Drinker, who is Professor of Industrial Hygiene at Harvard. Doctor Drinker.

BY DOCTOR DRINKER:

Doctor Vorwald, Ladies and Gentlemen: I'd like to make it clear to those whom I don't happen to know personally that I am not a physician and if questions should, by any chance, come my way which are medical, I take second

place to no one in the matter of evasion and I will evade them and pass them along to somebody else.

The first paper this morning is by Mr. Urban of the Laboratory here, on Industrial Hygiene Studies in Coal Miners in Two Geographical Areas in the United States.

BY MR. URBAN:

Coal is a sedimentary rock which, because of its high energy value, has been and is of outstanding importance to man. Coals occur in all geological ages since the Devonian period about three hundred million years ago. World-wide distributions of coal originated in the _____ and in the _____ periods and in the later tertiary period, most of the lignites or low ranking coals of the world were formed.

The extensive distribution of the individual coal seams implies swamp accumulation on broad deltas, coastal plains and broad interior low lands where shallow waters rest throughout the year. All ordinary coals are of vegetable origin. In the sedimentary cycle of coal, the immediate source of materials were the prolific plant tree growths and the carbon dioxide which was drawn from the surrounding atmosphere and surface waters.

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The trees and plants which fell into the swamp waters underwent partial decay and the residue accumulated on the bottom of the swamps to form peat, which is the first

stage of coal, all coals. It has been estimated that from 125 to 150 years of time of accumulation were required to produce the equivalent of one foot of bituminous coal. From 175 to 200 years were required to produce one foot of anthracite coal. An essential to coal formations is the arrest of bacterial decay in the submerged vegetable matter before complete destruction takes place, so that there is some residue to accumulate. This is brought about from the decay-promoting bacteria to render the water toxic to themselves.

The type of coal eventually formed depends on the environment, the kind of plants and particularly on the duration of the bacteria decomposition. May we have the first slide, please?

In non-technical terminology, four main types of coal are recognized, anthracite or hard coal, bituminous or soft coal, canel, which is actually a special type of bituminous coal, and lignite. These types may be further subdivided into ranks. Each rank designates coal of specific energy values and chemical and physical properties, semi-anthracite, intermediate coal between anthracite and bituminous; there are actually five different kinds of bituminous -- sub-bituminous and brown coal are intermediate between bituminous and lignite, andhracite being termed as the coal of the highest type and lignite the lowest type.

Peat is a fuel. It's not a coal, but a mixture

of partly decomposed vegetable matter in which bacterial action, for the most part, has been interrupted. Subsequent physical and chemical changes produce coal of the above ranks. Where these changes have reached completeness, graphite is the result.

Physically -- rather, chemically, coals contain hydrogen, carbon, oxygen and impurities either in simple elements or in complex combinations. The chemical changes which occur when the higher rank coals are formed from peat are the progressive elimination of water, oxygen and bituminous - either the solid and semi-solid hydrocarbons, the increase in carbon as fixed carbon in volatile matter, the consolidation of hydrogen development, heavy hydrocarbons, and an increased resistance to solvents, oxidation and heat.

Some of the physical changes which may occur in coal formed from peat, in the highest ranks, concern the by-products of those coals of the higher ranks which contain lignose, gums, waxes, oils, resins and fats. Those resins and waxes which are less assailable by bacterial action are present in most coals.

Coal beds occur within so-called coal regions which consist of alternating beds varying in thickness, of sandstone, shale, clay, almost -- mostly of fresh water origin. A coal measure generally contains several coal seams separated by sedimentary depositions of these various

minerals.

In Pennsylvania, for example, there are 29 coal seams aggregating 106 feet of coal with beds varying in thickness from a mere film up to 100 feet. The famous Maimot seam is fifty to sixty feet thick. In Alabama, there are fifty-five coal seams. In England, the aggregate thickness of coal is 85 feet and in Germany 120 feet.

Now, during the past several years, the Saranac Laboratory has conducted industrial hygiene surveys in a number of underground coal mines located in various coal fields throughout the eastern section of this country. Data from such - from six such surveys has been selected for presentation. Each of these mines extracted a bituminous or soft coal from a different coal seam, with the exception of two mines, five and six which I'll show you later which worked in opposite ends of the same seam.

They ranked in size from employment of about 300 to 1,000 men. The deepest mine was about 600 feet below surface. All of the mines were entered either through vertical shafts or inclined slopes. Modern methods of mechanized mining were followed in each case, utilizing both track and trackless type of equipment. A limited amount of hand work, hand shovelling into conveyor pans was also done.

All the mines were generally regarded as gaseous.

Considerable attention in each case was, therefore, given to safety, to ventilation and dust control. Now, for these reasons, data from the - from these six mines can be compared.

It is our further belief that some of the conclusions derived from these studies can be employed also through other coal mines, even coal mines of the anthracite type. Our primary concern in these surveys was for hazards of a respiratory significance. The nature and effect upon man of the gasses commonly encountered in bituminous coal mines, gasses like methane, carbon dioxide, carbon monoxide and oxygen-deficient air are rather well established. Our investigations were directed mainly, therefore, to a study of the air-borne dust, to which the workers were exposed.

May we have the first slide, please? Now, early in our investigations, it became apparent that a number of different minerals would be present in varying proportions throughout the same coal seam. A method was developed by us for the collection of air-borne dusts for chemical analyses. It consisted of a small one half horsepower United States Bureau of Mines approved suction motor set, connected to a paper filter by a two-inch diameter smooth ball hose. The paper was enclosed in a holder which was elevated to breathing level height by a tripod. The apparatus was completely portable and was used wherever electric power was

available.

In operation, dust-laden air is drawn into the filter. The dust is retained and the clean air passed on through to the fan. This is your fan motor set; this is your hose; this is your filter paper holder. We just set it on an ordinary engineer's tripod so that we can carry it around at the same time keep it up in the air, and the filter paper holder section is here. It was a simple metal affair. It had a screw cap on it and the filter paper was held in position there tightly.

Now, we're not sure about the efficiency of collection of this device, although we know it's very high. Through experience, we have learned, for example, that we can run this thing all day in a coal mine, collection a -- collect a deposition on the filter which would be on this side, heavy enough to completely fill up all the cleats, and yet when we take the filter paper off, the back of the filter - the color of the back of the filter hasn't changed at all, that is if any particles did pass through, there were very few of them, so that the paper must be an efficient collecting device.

The device was employed for collecting the air-borne dusts from coal face operations, from workings in rock and during the coal train haulage. Figure two illustrates a typical section through a coal formation of two of the

mines surveyed. This particular bed was the deepest of three coal seams which were being mined at the time in the same coal field or coal measure. In other words, three operators, three other beds had different elevations in the same geographical area and were being mined; this just happened to be the deepest.

The roof above the coal bed was a mass of sandstone deposits separated from the coal by a narrow layer of top slate. Lying beneath the coal were deposits of other sediments as coal slate, fire clay and sandstone. In addition the coal bed itself contained several other sedimentary depositions, termed locally as binder seams. This is your main sandstone area roof. We have a top slate layer here, some mines this wasn't present and others it was present. Some places, geographically, we called it - in some places it was called broceli.

This is your coal face or coal bed. Here are other sedimentary depositions within the coal face itself or rather inclusions of sedimentary materials. Actually, while we consider that this is mined as a single coal bed, actually, we have one, two, three, four coal beds representing different sedimentary cycles of coal formation, because each one of these binder seams was lain down after the lower coal seam was deposited.

In the other coal mine study, the respective coal

seams were similarly enclosed by sedimentary deposits of varying thickness and mineralogical composition. In some coal beds, the binder seams were less prominent; in others, however, the coal seams included, in addition, deposits of Golden Bright, again these local terms, and rock layers as much as eight layers in thickness.

In the mine represented by this coal face, bulk samples of each of the various rock horizons, both enclosing the coal and included in the seam, were taken for chemical analyses. All contained free silica.

In the sandstone, the analysis, the average analysis of several samples showed it contained sixty-seven percent free silica, the top slate twenty-one and a half, the binder material which again was a sedimentary deposit, probably a shale material, contained forty percent free silica. The coal itself was taken very - quite a number of samples - and the average of these samples was only three tenths of one percent free silica. Coal slate and slate - the two in this particular case were so intermixed that we couldn't separate them and the combined analysis, the combined analysis showed thirty-one point one percent, the fire clay 39.2 percent silica, the sandstone similar to that, so all of these sedimentary depositions other than coal contained appreciable amounts of free silica.

Chanel samples were obtained from across the

entire coal face from the roof to bottom and showed free silica samples ranking from 1.5 to 7.2 percent. The average of thirty samples was 8.3 percent silica. The air-borne dust created by all operations during the complete day's shift was collected on filter paper. The free silica values of these samples, however, ranged from three tenths of one percent to seven tenths of one percent with an average of five tenths percent.

The chanel sample was simply a run or cut of the roof of uniform size directly across from the top to the bottom and collecting all the droppings that came out of that room and analyzing those for silica. These results indicate that although silicious materials were associated with the coal bed, most of the dusts created by the coal face operations originated from the coal itself.

- We can see that pretty well even though we do have the silica-bearing materials here, the air-borne dust only contains half of one percent free silica.

May I have the next slide, please? To illustrate this point, still further, table Two shows a comparison of the petrographic analyses of three different types of sampling obtained from the same coal face. A quartz content of the coal only in this particular case was a hundred to one percent. A chanel sample from the entire coal face showed three percent silica and a filter paper collection of air-borne

dust from all coal operations revealed but three tenths of one percent silica. When this first sample was obtained, we instructed the man that was doing the drilling with a drill, to drill into as clean a coal area as he possibly could and then we collected all the dust from the drilling and the quartz content of that dust was a hundred and one percent, -- rather, one hundredth of one percent.

Undoubtedly, for the proper evaluation of the silica hazard in coal mining, analyses should be made of the air-borne dusts rather than of the parent materials.

Dust conditions to which men are exposed for coal mining can be divided for study into three general categories: The dust divisions related to operations of the coal face, those existing in coal working sections which are adjacent to the coal face and those present in other areas of the mine, through which men must travel or in which general work is done.

May we have the next slide, please? Table 1 shows the average concentration of dustiness associated with common coal face operations in six bituminous mines. 250 samples are summarized in this table. One of the first standards for permissible levels of dustiness for silica, applied in this country, judged that if in any given dust concentration less than five million particles per cubic foot of silica were present, the condition was considered

safe. Recently, the United States Conference of Industrial Hygienists have proposed that no permissible concentration of any dust should be denser than fifty million particles per cubic foot. Following this, the United States Bureau of Mines suggested a maximum weighted average dust level for coal mines of twenty million particles per cubic foot based on a work shift exposure.

Now, the first standard has some experimental basis. The other two, in part, are attempts to establish standards of good practice. In mines No. 1, 2, 3 and 4, all the coal face operations where wet methods were used, the dust conditions were probably with one exception, complied with, or they complied with all of the above standards. That exception is this mine - this 94.0 figure. These dust counts are all in millions of particles with the particles being all less than ten micron per cubic foot, so this 94.0 figure, if it was properly weighted by the time required to drill the five or six holes in the face, which ordinarily was about a half an hour, it was probably weighted against the number of times that the man had to drill these holes in an eight-hour shift, would probably be less than fifty million particles per cubic foot, probably closer to that.

No air-borne dust samples were collected in these first four mines. We collected bulk samples but

not air-borne samples. From other observations, however, from a comparison of bulk samples from one, two, three and four mines, with five and six mines where we had air-borne sampling, the silica content was probably but slightly higher than that reported in five or found in five and six.

Now, dusty conditions were experienced in Mine Number Five and still higher dust conditions were found in Mine Six, even when using water. Based on silica content, however, the highest count taken was 3.3 million particles per cubic foot of quartz, that is even this figure of 672.3 million, with cutting dry, Mine Number six, that's total dust - the free silica count in that was only 3.3 million particles per cubic foot.

In Mine five, and particularly Mine Six, the coal was much more gracile, it was much more dry and gave rise to more dust than did the coals of any of the other mines studied. These results indicate that in most bituminous coal mines, dust conditions of good order can be maintained. In exceptional cases, however, additional precautions are required to prevent dust levels from approaching a silica hazard.

You can see in this dust count, this dry operation of cutting went up two hundred million particles higher, that we would approach an equivalent of fifty - of five million particles of silica present. We would be introducing

a silica hazard simply from the silica which was present even though at low concentration.

Table three, please. The next slide, please. Levels of dustiness at coal section operations other than those at the coal face are shown in Table 3. In Mines 1, 2 and 3 and 4, in this case also Mine 5, conditions were maintained in good order; although there were - they were moderately high in Mine 6, the silica count was low.

By shunting car cycle, we mean that we went - we travelled with the operator of this particular car. The car itself represents a - looks like a large shallow pan on four wheels, and he travels from the loading station in the coal face to the dumping station into the mine cars through the - through the coal section itself, and our sample was taken during the course of this complete cycle. When the shuttle car arrives at the dumping station into the coal cars, the coal was unloaded.

This particular operation gave rise to, in some instances, to more dust than in other parts of the operation of the shuttle car cycle. You can see here 127.8 for that particular operation alone; that 127.8 is part of this 163.5. The general air in the roadways was pretty clean although in Number 6 mine, conditions were ordinarily dusty throughout, and also dusty in the roadways.

The next slide, please. Dust counts taken in

intake gas supplies to the six mines showed consistently low values. It is in some of these airways that men are transported to and from their work places and where coal car haulage is done. In some of the mines, sand was used on the tracks to give the locomotives additional traction.

Several air-borne dust samples were collected on filter paper in these mines, from the position of the motor man during the course of an entire shift's operation. The levels of total dust and of silica in each instance were within permissible limits.

This (next slide) represents the area in the mine frequently travelled, where car haulage is done and the area of the mine where the work - where the workers spend possibly an hour or an hour and a half of his day just in the course of transportation.

The next slide, please. Table five is a summary obtained by the United - obtained by the U. S. Bureau of Mines in sampling the roof blocks common to fifty-one coal mines in nine eastern states. It was presented by James Westfield at the American Mining Congress meeting in 1951. It shows that more than ninety percent of the roof blocks in these mines were shale and sandstone, averaging 26.5 and 55 percent free silica respectively. These values are in accord with the findings of our survey.

Unquestionably, a definite silica hazard exists

in coal mines when work is done in rocks other than coal. Shafts or inclined slopes are driven at times which required penetration in the rock. In some mines, rock falls and falls of roof rock must be removed in the course of operation. Roof folding is a new practice which necessitates the drilling of deep holes in rock. When such work is done dry, resulting dust levels are high.

It has been demonstrated conclusively, however, in other rock mines and in tunnel work, that levels of permissible dustiness can be maintained by the proper use of water and ventilation. Some of the coal mine surveys follow this practice. Other mines, although they drill dry and work in rock dry, provide the men with filter respiratory equipment.

In conclusion, it must be remembered that these methods are in respect to modern day practices of mining. It can be applied only as changes and practices remain essentially unchanged. In any occupation, the occupational dust conditions to the exposed worker, the history of the mine as well as the history of the man must be taken into consideration. Disease conditions may well be the end results of dust exposures which he has experienced previously. Thank you. (Applause).

BY DOCTOR DRINKER:

We will try to limit the discussion on this paper

to about five minutes, because we're a little bit behind time. I'd like to ask Mr. Urban one question. Are you bothered by the bits of paper interfering with those samples in petrographic analysis when you use that completed filter?

BY MR. URBAN:

No, we have pretty good success with it and Doctor Durkan is here, I think, and can tell you about it. We have analyzed very small papers successfully.

BY DOCTOR DRINKER:

Are those the filters that are used in the Chemical Warfare Service Mass classification, you get it from the M. S. A.?

BY MR. URBAN:

I'm not sure about that, get it from M. S. A.

BY DOCTOR DRINKER:

That's the type we use.

BY MR. URBAN:

The only difficulty we had was trying to sample fields that were a little high in relative humidity, the paper getting wet, but where the humidity was less than say ninety percent, we didn't have too much trouble.

BY DOCTOR DRINKER:

Doctor Fletcher?

BY DOCTOR FLETCHER:

I'd just like to ask one or two questions, make

one or two comments on this most interesting paper. First, as to the method you use for free silica analysis. We have recently in Great Britain, circulated a sample of dust around about six laboratories, and using three methods of free silica analysis, and the answers ranged on the free samples of dust from about one percent to ten percent on the same sample of dust sent around to different laboratories, so I think the free silica figures are very significant in relation to the method used, and also even to the individual using the method.

Secondly, I notice you did petrographic analysis of air-borne dust. Now, that has eluded us so far. We have not been able to study inhalable dust under five microns in diameter by petrographic analysis so far. The petrologist will go down to about ten, fifteen microns; below that, he says, 'Sorry, can't help you'. We hope we're getting somewhere by a new method in which the particles are crushed under the microscope, into a thin sheet and we may be able to identify them petrographically that way; we hope so.

Thirdly, I notice you took samples on filter paper; you apparently dispersed them to get your counts. Of course, the man doesn't do that. He breathes the dust in the air, and his own system and his respiratory tracts takes the particles, and our view at the moment is that he doesn't inhale anything over a particle size with a terminal

velocity equivalent to a particle of - a spherical particle of unit density about five microns diameter.

Now, of course, if you collect dust on a filter and then disperse it, you break up all the aggregates and the aggregates are, of course, the big factor in the actual inhalable dust concentration in any coal mine, and I wondered what your views were on that point.

And, lastly, I see you quoted average dust concentrations without mentioning any standard areas. We find that the co-efficient variation of precipitate amounts, I quote from memory, it's of the order of fifty percent. There is a colossal variation from moment to moment, and just an average dust concentration taken regardless, to the moment at which the man is working and without reference to the various - the variability we feel may be highly misleading, and I wondered what your views were on that question.

BY MR. URBAN:

I'm sorry I didn't make myself clear. The dust counts that we made, the dust counts that we made were not on filter paper but we made them with the standard United States Bureau of Mines Light Field count. That is we actually sampled atmospheres and counted particles less than ten microns. The filter paper collection was simply to collect air-borne dust.

Now, it's true that, petrographically, we can not

see, that is we can not analyze petrographically, particles lower than five microns. In our case at the laboratory, we employed three men. We used topography, chemistry and index, and X-ray defraction combined to give us some idea of the mineralogical components of the dust. We had done the air-borne samples by petrography, so we could get our other mineralogical components, and their action, like calcium, magnesium, iron. The quarts in that table was done chemically.

Now, the reported average counts for several of these: One was a very practical one. If we put the ranges on that table, there just would be so many figures we could not recognize the table, and certainly we did find variations as much as fifty percent from one count to another in the series, but we did report the average because we studied the series. The average looked fair and we reported the average.

BY DOCTOR DRINKER:

We're doing petrographic analysis, Doctor Fletcher, on particles that are well under five microns. The only one you've got is past history. It seems to me it's perfectly analogous to what you demand in radiological analysis. You won't give a positive diagnosis of silicosis unless you know the man's history.

Well, I don't see - this seems to me perfectly

analogous, if you know the past history of the dust and know it accurately, you can do things petrographically that you can't possibly do if you come into an unknown, and following that argument out, it's common knowledge now that a number of our laboratories in the United States do do petrographic analysis on dust well below five microns. They wouldn't teach you that in a course in petrography, but it's being done.

I asked him that designedly, that question about the interference of the fibers on the filter, because Briscoe and Matthews and your other pals have said that you can't do it with those. My point is that the numbers of those fibers are so trivial that they're not of any consequence, but I'm sure that the Americans Don't profess that their accuracy in these determinations in any way is remarkable, and we have just as bad troubles on the big ranges of concentration as you do.

The next paper is by Doctor Fletcher on Radiological Classifications.

BY DOCTOR FLETCHER:

Mr. Chairman and Members of the Symposium: I'm sorry that I should appear again so shortly. I hope that there are not too many radiologists in the Symposium this morning, and although I should like there to be in some ways,

BY DOCTOR FLETCHER:

Mr. Chairman and Members of the Symposium: I'm sorry that I should appear again so shortly. I hope that there are not too many radiologists in the Symposium this morning, and although I should like there to be in some ways,

and that is that I find that radiologists known as little about radiographs of pneumoconiosis as general physicians know about the clinical and physiological picture of pneumoconiosis.

Seven years ago, when I started my work in relation to pneumoconiosis in South Wales, I consulted a very eminent radiologist in London and he told me that the radiological side of the pneumoconioses had been completely worked out and there was no further cause to do any work in that subject at all. And when I got down to South Wales, I started studying literature.

Could I have the first slide, please? And this is a brief summary of the sort of thing I found about the systems of classification that had been widely used in relation to coal miners, particularly in various countries. There was the original ILO classification with the three stages which were partly radiological and partly clinical; there was the Miners South African Classification that some author~~s~~ had attempted to apply to coal miners with all these elaborate classifications here, mostly based on the size of the operation; the American Public Health Service classification used in those excellent reports in the hard and soft coal miners, again with various elaborate subdivisions.

Now, these ones here, and this side of the diagram, I know the real equivalents in these classifications, because

we've done actual readings of the same sets of films in the different classifications with the people using them, and we know something about the equivalents.

Over here, the equivalents are a bit different. We have the French one, which is normal with a little subgroup of normal, the 'pathologique normale' which is a rather nice bit of French compromise, the little subdivision of normal, and the fibrotic F here, 'Fibros modulaire', and 'supernaturale', and these were based on the size of shadows.

The Germans, in deference to them, I have simply - I have simplified what they actually do. They actually put in the intermediate classifications of one and two, two to three, between two and three, and they even have a subdivision here, naught to one to one, and naught to one to one in between these two, and then the Silicosis Boards in England have modified this original one here. Genhardt, who wrote those, did the original survey work. In South Wales they had done an original classification of their own, using a term reticulation, which I regard as an abomination, because it is being used by different authors to indicate anything from a normal film up to the most advanced nodulation and then that was the situation, and we eventually worked out a new system of our own which I have indicated roughly here.

Now, the three reasons why we wanted a new system. First of all, none of the existing systems recognized the basic distinction we wanted to make between radiographs that showed only generally discreet nodular shadows and those that had, in addition, local, coalescent or massive shadows.

Our reason for wanting to make this distinction was largely based on Professor Goffe's work, as he made a very clear pathological distinction between what he called simple pneumoconiosis and massive fibrosis, and I'll show that in a moment.

The second thing was that in our follow-up studies of radiographs of men progressing, we found that the progression of the disease in those with generalized discreet shadows, was manifested always by an increase in the number and profusion of shadows and only in proportion by an increase in their size.

Now, all the other classifications indicated progression by increase in size, ignoring increase in profusion.

And, the third reason was that we wanted to make our classification purely radiological. We didn't want to have any confusion with clinical symptoms which had appeared in some of the others.

The next slide, please, shows this basic distinction -- the radiology -- between coal deposits in the lung --

next slide -- and those with, in addition, large massive shadows, and that is the basic distinction that we make in our classification.

Could I, just for one moment, have the first slide back again. I'm sorry to go out of order, but it just would help if we could have it back. Well, now, we devised a system which has been published and which some of you know about and we advocated a similar system at the ILO Conference at -- I say we did -- I advocated a similar system at the ILO Conference in Sydney in 1950, and somehow or other, they suggested a system of classification that bears rather a resemblance to the one that we have been using.

Later on, we thought that the just mere announcement, by an International Conference of a system of classification that was a good one wouldn't do much to increase international understanding. In any case, the ILO have not yet published the report of this Conference. Things go slowly in Geneva. The Conference was over two years ago, but nothing has appeared yet about it, and we wanted to be able to understand epidemiological results that were being obtained by workers in coal fields in the north of France and in the Ruhr in Germany, and we, therefore, sent them sets of films from our mines and asked them to send us sets of films from their mines.

We carried out readings in their classification

and our classification, and we undertook meetings to discuss this and eventually, both in France and Germany, they have agreed to use this type of classification for their work, but have introduced a small modification that they want to distinguish in the groups of simple pneumoconiosis, that here the type of films with very fine capacities, moderate size and larger size, so as to retain something of the original indication of size, but in a secondary role.

Well, now, the actual classification used, consists of a normal group, three subdivisions of discreet shadows. We originally had four; we have dropped the fourth now. Three are quite sufficient, and then they have four divisions of cases with massive shadows.

Now, the next slide. Here is a radiograph. It's almost impossible to demonstrate these things with lantern slides, but I have got quite a collection of films here if anybody would like to come and see some of the original films afterwards, but in classification 1, we demand presence of minute opacities, usually one millimeter to 1.5 millimeter diameter, in little clusters in at least two rib spaces, each area occupying an area of one square centimeter.

In Classification 2 -- next slide -- we require that these opacities be distributed throughout both lung fields, but they are usually sparse at the periphery and up at the apex.

By Category 3, they have to become generalized by all lung fields and are now profuse and extend right out to the periphery.

That is the basic classification of the simple pneumoconiosis, but in addition, we do recognize these different types, that is the mixed type which we call mixed, or 'Micron Nodulaire' which also begins with 'M', and the Germans call it _____, but unfortunately, we're prepared to call it some other word which I forget at the moment which does begin with an 'M', but -- next slide -- there is a common type occupying about ten percent opacity in simple pneumoconiosis, in which these little minute opacities occur in pure culture which we call the pinhead type; the French have had the courtesy to call it 'Pinhead Fleshaire'. The Germans - the Germans have insisted on calling it _____, which is a little different, but anyway it indicates this pinhead period.

On the next slide, we have, on the other hand, the nodular type of film, in -- fortunately, 'Nodulaire' in French, and 'Nodular' in German, and we indicate that with an 'N', following the number indicating the category of the simple pneumoconiosis, and this, of course, the nodular appearance tends to occur in men with a heavy rock exposure who have actually got classical silicosis, together with coal deposits, the only condition which justifies the term 'Anthracosis-silicosis'.

Well, now, coming on from that -- next slide - we come to the beginning of what we call progressive, massive fibrosis, PMF, which the ILO calls pneumoconiosis, with coalescent massive shadows where, in addition to the general condition, you have an area with larger opacities, amounting to rather more in diameter than appear, and although, in many films, the radiograph also shows a definite mass. That is the beginning which we call PMF, to distinguish the categories from the numbers we use in simple pneumoconiosis.

The next slide. These cases have the peculiar property that they tend to progress whether or not the man is exposed to further dust inhalation. Our simple pneumoconiosis, we see progression from Category One to Two or Two to Three only under conditions of dust exposure where presumably further coal dusts are being laid down in the lung, but here is a radiograph of a man who leaves with a shadow, leaving the mine in 1941.

Next slide. By 1943, he has now a sizeable mass of it here and another one beginning there -- (next slide) -- and in 1947, he has a very definite mass here. Note the next one. The next one -- may we have the next one -- this is 1947 with now a large mass in both sides of the lung field, and we indicate these decreasing size of masses with the letters B and C, B for shadows that extend only over three anterior rib spaces, C if they extend over more than

the three anterior rib spaces, just to give an indication of their size. That's not a very important distinction, so far as we know.

And, lastly, we use the Category D -- next slide -- for those which, in addition to massive shadows, have gross distortion of the diaphragms, the mediastina here, and with emphysema at the basis, which, as this radiograph shows, can be quite extreme in many of these cases.

So, in summary now, -- next slide -- we have the classification slide which I have used both in France and Germany; that's why it's international - in which we have the normal films with naught and the French again wanted to retain some vestige of their pathologique which we call X, which is a film which you can not put your hand on our heart and say it's normal because there is something that suggests pneumoconiosis to you, because it might well be something else, some sort of non-specific fibrosis, emphysema or what have you, bronchitis; and so, if you like, you can call it X. We regard it as pretty bad form in the pneumoconioses to use X; we only do it when we're absolutely forced to, because we just can't make up our minds.

Then, we come on to simple pneumoconiosis, which is three categories depending chiefly on the diffusion of the opacities, on their diffusion up to the rib area, with the pinhead, micro-nodular, mostly put in for the consideration

of the French, and for description, but we don't regard these categories as having any great significance. Natural history, and the prognosis and everything else, of the pinhead, micro-nodular and nodular is similar.

Then, we come to progressive fibrosis, A, B, C and D, depending on the size of the shadows, and eventually D, which you can't see on the table there which has distortion.

Well, now, that is the classification, and now I want to say one or two words to try and persuade you that it may be quite a good one. First of all, it is a purely radiological classification. It is used by looking at the radiograph and without any further information at all, which is what a radiological classification should be.

Secondly, the categories are based on studies of the progression of the disease, so that the earlier categories represent earlier stages in the natural development of the disease, than do the later categories.

Thirdly, in justification of our categories of simple pneumoconiosis, we have found evidence which I shall show in my next talk on the epidemiological side, evidence that there is a fairly direct quantitative relationship between the amount of dust exposure required to produce these three categories. The ratio of dust exposure required to produce Category Two is about twice that required to produce

Category One, and to produce Category Three is half as much again. The actual ratios are about six to ten to fifteen. So that these categories do represent a fairly direct quantitative relationship to the amount of dust inhaled. We want to confirm that by chemical analyses of the amount of dust in the lungs in these categories but we haven't yet had the results of that completed.

With regard to the relationship to the underlying pathology, there is difficulty. We, as radiologists, on the radiological side, think the difficulty lies on the pathological side and the difficulty I referred to yesterday, and that is the difficulty of getting the pathologist to be quantitative and to say just how much abnormality there is in different lungs, but we have got Professor Goffe and his colleague, Doctor James, to classify a large number of lung sections done by his technique, which we had radiographed too under three systems, one according to the profusion of the opacities, and also according to the severity of the focal emphysema.

There is no relationship between our categories and the severity of focal emphysema, none. The profusion of the opacities is not effected by the development of focal emphysema, but there is a relationship, a definitely rough relationship between our categories and the dust foci, which is some indication of the amount of coal in the lung.

Lastly, the relationship to disability is of great interest, but as Doctor Hugh-Jones is devoting his talk to that subject, I will leave it to him, and I will only say here that there is little relationship to disability here, but a very great justification of this distinction between simple and massive fibrosis, on disability grounds.

Now, one point, I don't expect any of you could see any abnormality in that film I showed you of Category 1. Well, now, quite experienced radiologists in England and in this country, are having the privilege of discussing some films of ours with Doctor Eugene Pendergrass in Philadelphia, and he expressed a firm idea that films of Category 1 and 2 were absolutely normal and he could parallel them with films of any man walking the streets of Philadelphia.

I tried to get him to look at these specific opacities which I thought justified the diagnosis. He said he was looking at them but even so we couldn't agree and so either we were wrong or Philadelphia was much smokier and dustier than it looked when I was there, or he was wrong.

Well, I was very worried about this and I was worried, too, about radiologists who said that they couldn't see any abnormality in our Category 1 and, as this Category is one which we have used extensively in our epidemiological work, it was of vital importance to see whether it was true

or not, so what we did was to collect 125 films of non-miners, men who had had no significant dust exposure. Some of them were members of our own unit, but the majority were men working in a power station on non-dusty occupations where we had a survey, so we got good films there. We mixed them up and ran them out with films of seventy-five miners of neighboring mines which had previously been read by readers A and B on survey, fifty of them being normal and twenty-five of them being category 1.

Well, now, we thought we'd be clever and not only read category naught, one, two, but we thought we'd put in an extra category naught, with a cross in it, to indicate that although we thought it wasn't quite category one, to indicate that the film showed as if it was a miner's film, because we not only believed we could read category one, but something earlier. So, that's what we asked the readers to do.

The films were read, interestingly, by four readers, A, B, C and D, and then read together by A and B, and by C and D, and finally those films, on which we differed, were read in consultation by all four together, and here are the results for the non-miners and the miners.

In the non-miners, A and B -- miners, first of all -- were rather cautious under these experimental conditions. They didn't want to show they had been reading

one wrongly and you will see of the twenty-five films, they read as one - they read ten as one; now, I pushed them down to normal miner and non-miner. In so doing, they didn't read any pneumoconiosis in the non-miners and only read one case as showing, looking like a miner, but not being category 1. C and D was myself and Doctor Gilson. We were more courageous and we read up a little bit; we read thirty-eight out of these films as showing Category 1, one as 2, and about the same number, about thirty-six coming in the normal group, but in so doing, we had read four of the non-miners as having Category 1, four of the 125, and nine as being suspicious.

When the four of us met together to try and straighten this out, we pretty well hit off the original level of reading the miners' films and we never once agreed that there was evidence of pneumoconiosis. I asked that A and B try and re-read these films using their normal survey technique. Here they were put off by the danger of reading things that they were frightened about and they did try and read them exactly as they read the survey films, and they very nearly hit off the same level as on the survey and they never once read Category 1 in a non-miner. They weren't attempting, in that case, to read the suspicious.

Well, I conclude from that, that at any rate, we

can distinguish our Category 1 and that it does occur only very rarely in non-miners, and there only when we're over-reading. When we're reading as we normally do, a Category 1 is a thing which is sufficiently rare in a normal not to have occurred in this group of 125 non-miners. Just the same, the sort of danger of less specific reading, I did send these films to two Germans who agreed strongly on this abnormality and you will see they read on the miners, and no less than twenty and twenty-two in the non-miners, as beginning silicosis.

So I think this type of experiment, mixing up completely normal films with films of men exposed to dust, is a completely salutary experiment. I recommend it to anybody who is engaged in this sort of work.

One small point of interest, what with these cases that C and D, Doctor Gilson and I, read as showing some abnormality. I was interested to know if it had any relation to Age. So I have just plotted it out in relation to Age. You see that there is a very clear relationship between age and this abnormality, and I believe that it is possible that these older men aged forty-five to fifty had, in fact, been exposed to a certain amount of dust and smoke in and around Cardiff, and that what we were reading as early pneumoconiosis, may have been the earliest signs of dust accumulating in their lungs with age.

What is most important is that even our overreading of Category 1 never occurred in a man under thirty, and most of our active epidemiological work is concerned with men in that age group, so that I am quite satisfied that our Category 1 is an abnormality attributable to the inhalation of dust in the - at least in the younger age group, and nearly always so even in the older age groups.

Next, I just want to talk very quickly about the question of observation which has been pioneered in this country in relation to tuberculosis, but I think we have pioneered it in our country in relation to pneumoconiosis. The first experiment we did was to get 102 films of miners ranging from normal to nodulation in the original classification. We asked ten experts to classify them into normal, early, definite, certifiable pneumoconiosis, and nodulation.

We found that, of these ten experienced observers, in thirty films, opinions ranged from one observer to the other, from one to four or two to five, that is one quoted normal, another certifiable pneumoconiosis, another nodulation. For shattering results, I took that up to a very eminent radiologist in London. He said none of them were radiologists. He said he and another radiologist would agree a hundred percent on these films.

The silly ass tried it, and the difference between them was greater than between all ten, so -- this is very

important -- the number related as showing certifiable pneumoconiosis by these various observers, varied. One showed that four of 102 films showed enough pneumoconiosis for certification; another said that 29 were -- I mean normal. The number certifiable ranged from 9 to 53. These were experienced observers. The average consistency--each observer read the film twice -- was sixty percent; sixty percent of the films the observer did give the same opinion on the two occasions; the record was eighty-four.

The other thing was under-exposed films. Some of the films were classified higher than the hard films. Well, now, we have done a great deal of work on this subject and we have attempted to improve this situation, first of all, by accurate definitions of the categories in words; secondly, by setting up standard films exemplifying the categories, so instead of just putting a film up and saying, well, that looks like Category 2, you put up another film alongside which has been agreed to be Category 2 and see if it looks the same and, thirdly, we have adopted the method of reading shown to be so valuable by Duorshonn and his colleagues, of duplicate reading. Every film on our survey is read on two different occasions without any knowledge on the two occasions what the previous reading was and any film on which two opinions differ is given a third casting vote to get the final category.

The next slide just shows -- no, we'll skip this one, I think this is just showing the errors in relation to the diagnosis of pure math in this international set-up. We'll skip that one. Now, this is our latest result of our readers A and B who do all our survey reading showing that first reading of five hundred films taken at random from a large survey of five thousand and their second reading here, and any film which was read normal on the two occasions never goes to a third reading, but if no two agree on two readings we have a third deciding reading, and you can note the lack of divergence of opinion, which is relatively small.

Lastly, on the question of understanding, I have told you that we have been taking films around in France and Germany to get them read and here is at least the result of 150 films which we sent out, which we classified as thirty in Category 1; thirty-two; thirty - three, and we sent them to them and put them into the international classification, and into the German classification as presently used, and you will see that while there is difference in the minds of the observers here, there is, on the whole, agreement. If they were to have classified that set of films and given us a prevalence of pneumoconiosis based on that classification, it would have been very similar to the prevalence that we would have found if we had read those films. We could understand their statistics, but if they

did it in their own classification, there was some agreement between the categories here. You will see that our Category B films are pretty evenly distributed, and what we call Category A were distributed through the whole range. It would have been very difficult for us to have any accurate translation of that sort of classification into ours, even if we had known what the results of this experiment were going to be.

At the present, the existence of different sets or systems of classification in the different countries of the world, and the different centers, militates very seriously against scientific understanding of epidemiological work. This system of classification has now been adopted pretty well uniformly throughout Great Britain. As I said, it is being adopted in France and in Germany. A conference is being held in Paris to further its adoption in France, next - the end of this week, on this Saturday, and I think the time has come for some international body, the ILO, to start being active in this field and carry on this sort of work we have been doing on our own, by getting sets of films sent around to the various centers where work of this kind is being done so that we can see what the relationship between different forms of classification is, and if possible, obtain the consent of the different centers in this part of the globe, to use a classification similar to that which we

we are using, not identical, because I have no doubt that you will have a great contribution to make in suggesting valuable modifications, but at least that we should adhere to using a uniform system so that we can know what you're talking about and you can understand what we're talking about in our radiological work. (Applause).

BY DOCTOR DRINKER:

Any discussion of this paper? Doctor Richards?

BY DOCTOR RICHARDS:

I don't think we're too far apart. I think I could understand Doctor Fletcher as he went along with this, very well, and I don't think I have anything particularly to add.

BY DOCTOR ORENSTEIN:

Mr. Chairman, I would just like to pay a tribute to Doctor Fletcher's work from the point of view of the tactical worker in his field. To many of you, this may seem to be a supererogation of trying to establish a whole lot of categories, but it is a matter of the greatest importance that there should be an understanding in this matter, not only from the point of view of epidemiological survey but from the point of view of such countries where compensation is based on stages of the disease and is not true to form simply because a man has pneumoconiosis. He receives a certain amount if he has pneumoconiosis in a certain stage,

and a certain amount for a larger stage and so on, and because of that point of view and because of the great damage that was done to the compensation question, the certification question by the 1930 adoption of that one, two and three which you saw on the right-hand side of the scale, when an attempt was made to correlate the capacity, that is to say compensation, to stages of radiographic appearance, now, a lot of good has been done by Doctor Fletcher in his endeavors in this way in particular, where he demonstrated this at great length and I'll never forget the night we spent in a small office somewhere up on the top floor of a Sydney building, threshing out this question.

I also would like to pay a tribute to the objective work which he carried out in trying to discover the errors of his own interpretation and of others' interpretations. A lot has been said by radiologists, if we have long enough experience and enough correlation with post mortem findings, we can classify with great ease. That's quite untrue. They can not, and a lot more work will have to be done to make that clear.

I merely want to say this, Mr. Chairman so that somebody who has to work in this field would speak for the great benefit this work has conferred on the knowledge of pneumoconiosis.

BY DOCTOR DRINKER:

Thank you, Doctor.

BY DOCTOR VORWALD:

Mr. Chairman, I should like to join Doctor Orenstein in his tribute to Doctor Fletcher and we here are indeed fortunate to have him with us and to hear what he has to say. I don't think that we're too far apart. Of course, there is divergence of opinion with respect to Category 1 and Category 2. That is where we find the greatest difference.

Doctor Fletcher, I know, is keenly aware of the technical qualities of the film. He hasn't mentioned it; perhaps he will, the necessity for having good films of good technical quality in order to classify these films. Then, I should also like to ask Doctor Fletcher one question, does his classification pertain only to the coal miners' lung, to the coal worker, or does his classification pertain to all of pneumoconiosis?

BY DOCTOR DRINKER:

Will you answer that, Doctor Fletcher?

BY DOCTOR FLETCHER:

Thank you very much indeed, Doctor Vorwald, for raising these two points. I am afraid I was getting behind time and I didn't have time but, of course, the technique of the radiograph is of the greatest importance. We have shown that the average of category given by ten observers

to one film, or films of the same man taken on the same day, under-exposed and over-exposed, the average opinion may shift by one and a half categories easily, and it is absolutely essential to have really good technique and we have devoted a great deal of attention to that point in our own radiographic work.

We have now, in our mobile unit, not only an automatic exposure control mechanism which Seaman's in Germany have devised for their automatic, which is quite remarkable in the way in which it enables uniform exposure to be obtained of men of various chest thicknesses and with variations in main supply, but we now have also an automatic processing unit which puts the films through at exactly the right time and temperature and in that way, we have been able to reduce the number or percentage of films that we have to reject on our survey as technically satisfactory from about forty percent right down to two percent.

About the width of application of this classification, it has been worked out on coal miners and this work has been done on coal miners, which is the big subject problem we have in Great Britain, but I think that in principal, it is applicable to all the world's pneumoconioses which manifest themselves in discreet capacities going throughout the channels, that is to say coal pneumoconiosis, what Doctor Sander was calling sidero-silicosis, silicosis and so on.

I think the same principals can be applied.

The members of the Silicosis Boards in Great Britain who see men from all industries, tell me that they find this method of classification valid for at least ninety-five percent of all the cases they see. There are the odd cases which you'll say, I'm sorry we just can't fit this in. You've got to put it down as unclassified.

BY DOCTOR RICHARDS:

Mr. Chairman, I think while we're discussing this problem of terminology, I'd like you to call on Doctor Johnston, from a clinical standpoint to say a word to you.

BY DOCTOR JOHNSTON:

I have nothing to add. I just think that Doctor Fletcher, in all the arguments yesterday, Doctor Fletcher has probably established Doctor Sander's point that everybody has pneumoconiosis, the classifications one or two; even the farmers I'm certain we'll see, that in farmers who live in impure air all their life, so everybody must have pneumoconiosis.

BY DOCTOR DRINKER:

There are a fair number of persons in this room who are not good Roentgenologists then, and there is one definitely of that number I'd like to ask a layman's kind of a question. I have been steeped in the doctrine started, Pankos and Sampson and right on down to the present experts,

that these radiological pictures differ enormously with the geology to which these men are exposed and presumably, Doctor Fletcher's interests are largely in the Welch coal fields. How does this classification apply to say our hard rock miners in the Missouri area, Pendergrass certainly must have brought that up in talking with you.

BY DOCTOR FLETCHER:

I think that the classification would apply completely, but that our category 1 and 2 are a little earlier than Doctor Pendergrass would recognize. One of the difficulties is with these very early abnormalities, you have got to study the film extremely closely. We say ten inches is the right distance from the observer to the film. A greater distance than that you just can't see these early changes and I put up that film, that slide of the results of distinguishing normals from these category 1, to show that if you do that, you can distinguish and all I can say to Doctor Johnston is that if you will get these films of these farmers and send us a group, we will mix them up with some miners and get some readers who don't know what we've done to read them, and I'll bet he'll find that the miners have got more dust in their lungs and have got a higher proportion of Category 1 abnormality than have his farmers.

If, on the other hand, his farmers have got a great deal of Category 1 abnormality, all right, I agree with him,

they must have some pneumoconiosis, and Doctor Lanza, I think, would agree too that it might be called occupational.

BY DOCTOR VORWALD:

Well, Doctor Fletcher, there is no way in which Doctor Johnston can win, is there?

BY DOCTOR FLETCHER:

Yes, yes, he can, because we can get a third group of real -- in our country, there is very little dust in farming; in fact, none at all, and we could get a group such as these power station workers who also are not perhaps a dusty occupation as your farmers. I don't know. I have heard about the Dust Bowl, but I don't know how dusty this is, this occupation of farming over here, but we could certainly get a group of true normals in agricultural workers in our country or in office workers in New York; looked to me as if the atmosphere was pretty clear there, and we could say these are normals. If we diagnose abnormality in them, then clearly this Category 1 we're talking about is absolute nonsense in terms of coal workers pneumoconiosis.

BY DOCTOR JOHNSTON:

Phil, I don't want to labor this point; I happen to have a daughter who has lived in California all her life. She has never been on a farm, never done any work of any kind, there is no bituminous coal in California. I can vouch for that. She does have, however, some markedness that

might be classified under two of your films.

Now, I don't know how, what we do about such people. We can get into a hobby classification or a work classification and still they show this little stuff.

Now, I'm still getting back that we must adopt a point if we're going to go ahead with your classification, to get back to Doctor Emmett Sander here that everybody has pneumoconiosis.

BY DOCTOR DRINKER:

Doctor Sander?

BY DOCTOR SANDER:

I'd like to clarify that. I don't say that everyone has pneumoconiosis which is visualized by X-ray. We all have some pneumoconiosis by strict definition, but not visualized by X-ray. You can have, I'm satisfied, you can have a lot of anthracotic pigmentation and still have a perfectly normal chest film. I think we owe Doctor Fletcher a great debt of gratitude for this precise classification. I see no reason why we can't adopt it for almost every industry in this country. Even the diathermaceous earth can go into Category A, B and C; they miss Categories 1, 2 and 3, because they never are nodular, and I don't see any reason why we can't use it for almost any and every dusty industry in this country, *with the possible exception of Category 1.*

BY DOCTOR DRINKER:

Doctor Fletcher?

BY DOCTOR FLETCHER:

I do want to be absolutely clear about one point and that is that normal individuals in Great Britain, that we have examined, do not show the abnormality which we classify as Category 1, except perhaps in one or two percent of those over the age of fifty.

Now, I want to make that absolutely clear. I am quite convinced that this abnormality that we're classifying here as a specific change produced by dust. I won't say that the odd case of sarcoidosis or non-specific fibrosis, dermatosis, God knows what, can't mimic it, but that's absolutely important from the point of view of industrial medicine. The odd disease which effects one in a million of the population, we needn't worry about if we make mistakes there, but this condition is specifically for - as I'm convinced by our experiments, for the development of dust retention in the lungs, as shown by coal miners.

I'm very interested in Doctor Sander saying that some people may skip Category 1, 2 or 3. I think that happens to the pottery workers in our country. I have seen films there, which show a shadow in which the background showed no pneumoconiosis at all, and I think that can happen, but I think the principals can be applied, and I do want to make it quite clear, and I think perhaps some of the

slides I shall show you on the epidemiology of the disease, will persuade you that this condition does develop in relation to dust exposure.

BY DOCTOR VORWALD:

I should like merely to ask one more question and that is, Doctor Fletcher, you have classified now an individual as belonging to Category 1, had he had some respiratory disability. Are you ready to compensate him for inhalation of dust?

BY DOCTOR FLETCHER:

I have the good fortune to be a research worker not concerned with compensation, and it's entirely a question of the legislative definition. The practice of the medical boards in our country at the moment is not to recognize anything less than our Category 2. My own personal view is that if a man has worked in a coal mine and shows respiratory disability and has any evidence of dust retention, Category 1, it would be fairer to bias the scales in his favor and give him the benefit of the doubt and say that that disability could be due to pneumoconiosis, but I think that what Doctor Hugh-Jones will say this afternoon may throw a little further light on that problem.

BY DOCTOR JOHNSTON:

Doctor Fletcher, while you're still there, do you have any figures on your present employment classification?

DR. SANDER: I don't say that everybody has pneumoconiosis which is visualized by the X-ray. We all have some pneumoconiosis, by strict definition, but not something visualized by X-ray. One can have a lot of anthracotic pigmentation and still have a perfectly normal chest film. We are deeply indebted to Dr. Fletcher for this precise classification and I see no reason why we can't adopt it for almost every industry in this country. Even the diatomaceous earth cases can go into categories A, B, and C; they miss categories 1, 2, and 3 because the shadows are never nodular. I see no reason why we can't use the classification for almost any dusty industry in this country, *with the possible exception of category 1.*

DR. FLETCHER: I wish to make it clear that normal individuals in Great Britain whom we have examined do not show the abnormality we classify as category 1, except perhaps 1 or 2 per cent of persons older than 50 years. I am quite convinced that this abnormality which we are classifying is a specific change produced by dust. I won't say that the odd case of sarcoidosis or non-specific fibrosis can't mimic it but that is unimportant from the standpoint of industrial medicine. We needn't worry if we make a mistake about the odd disease which affects one in a million, but this condition is specifically associated with the development of dust retention in the lungs, as is shown by the coal miners.

I'm interested in Dr. Sander's statement that some persons may skip categories 1, 2, and 3. I believe that happens to pottery workers in our country and I have seen films supporting this belief.

DR. VORWALD: If you classify in category 1 an individual who has had some respiratory difficulty, are you ready to compensate him for the inhalation of dust?

BY DOCTOR FLETCHER:

I hang my head in shame and say that although we have been agitating in our units for information, for four or five years now, for pre-employment and regular examinations of coal miners, they have not yet taken place in Great Britain, but in those films I showed you of non-miners, some of those might have been applicants for coal miners and all I can say is we did not diagnose Category 1 in any man even when we were reading rather generally, we didn't diagnose it in any man under the age of thirty-five.

BY DOCTOR DRINKER:

The next paper is by Doctor Vorwald on the Pathology of Coal Miners Pneumoconiosis. Doctor Vorwald.

BY DOCTOR VORWALD:

Mr. Chairman, Ladies and Gentlemen, at the onset, I wish to say that we, in planning for this day's session, made every effort to bring Doctor ^{Goffe} Goffe from England. As you know, Doctor Goffe is Professor of Pathology and Bacteriology at the Welch National School of Medicine in Wales and he has had perhaps more experience with the pathology of pulmonary changes in coal workers than any man, and it is unfortunate that he could not be with us.

Now, in a general way, our concepts of the changes in the lungs of coal workers which have come to us here in the Saranac Laboratory for study, conform in general with

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Now, in a general way, our concepts of the changes in the lungs of coal workers which have come to us here in the Saranac Laboratory for study, conform in general with

the concepts expressed by Doctor Goffe. In some instances, however, there is divergence of views which I hasten to say is occasioned perhaps by the fact that our study of lungs of coal workers has been limited to relatively few cases. In addition, it is possible that the conditions of exposure experienced by coal workers in this country are different from conditions in Wales.

Furthermore, the problems pertaining to the inhalation of coal dust are indeed complex. Now, that complexity may be more fully appreciated should an attempt be made to answer the question as to what is coal dust, and then may I have the first slide.

Now, this slide, taken from a publication by Parkers in Coal Science, by Doctor Banion, Director of Research Laboratories, the British Coal Research Association, here Doctor Banion has tabulated some of the various rare elements in coal ashes.

Now, the data, as we see them here, are expressed in grams per ton or parts per million. To give an approximate idea of the concentration factor, comparison may be made with the average amount of the same element found in the ordinary rocks of the earth's surface as we'll use this factor. As for Arsenic, As, arsenic is one thousand six hundred times - one thousand six hundred times more concentrated in the ash of certain coals than in the average rocks

of the earth's surface, so we can also go through the listing of the other elements, beryllium, germanium, lead, nickel, platinum, zinc, zirconium, and you can multiply, you can see the factor, the concentration of those substances in coal ash.

Now, it is possible that some of these substances exist, per se, in coal in the dust breathed by these individuals and it is also known that some of these substances produce damage to the lungs.

In attempting to define what is coal dust -- next slide -- the problem becomes even more complicated should we examine the so-called coal tree, and here one visualizes the production obtained from coal by carbonization. Now, many of these products may exert their effect when coal dust becomes deposited in the lung. I am not too clear about this, but I merely present it to give exposé to the possibilities at least that some of the complicated features as we have seen them, pertaining to the inhalation, deposition of coal dust in the lung, may be related in some way to all these products which are derived from coal.

Now, what are the changes that occur in the lungs of some coal workers exposed to coal dust? This is the chest Roentgenogram of a 38-year old, I mean of a fifty-year old white male, who has worked for 38 years as a motor-man in a bituminous mine. Now, his medical history was i

irrelevant except for two years prior to death when this individual suffered from dyspnea, from exertion, I mean, especially on exertion, and from productive cough. Furthermore, he had clinically manifest arterial sclerosis, which - and ultimately developed multiple infarcts in the lung and brain and he died in coma.

The X-rays, as you see here, the lung markings are slightly exaggerated. There is a primary complex, as you see here, in the right lung. There is no definite Roentgenographic evidence of change, comparable at least with a diagnosis of silicosis. The gross section of this lung exhibits diffuse pigmentation, slight focal emphysema, as you see, rather coarse in type, some focal emphysema in the lower lobe of this lung which is of the right side. The lymph nodes are small, but they are slightly pigmented, and this is the section of this lung and it happens to be the left upper lobe, and we find that there is focal deposition of dust scattered everywhere throughout the lung. Furthermore, there is also deposition along the vascular fronts which gives rise to what we call a linear deposition. Also one sees the dust deposited in the tissue immediately below the pleura. Furthermore, one can see at least the disproportion between the size of the alveolar spaces which we identify as the anatomical evidence of emphysema, also note the tracho-bronchial lymph nodes which are only slightly

enlarged and pigmented.

This is from the right upper lung, right upper lobe. Now, in addition to having the focal deposition of dust, the linear deposition of dust along the vascular tracts, we also have a large area of deposition of dust and in addition, the emphysema in this instance is quite obvious, occupying one small portion of this lobe, but it is also apparent in the mid-portions of the lung and quite well scattered throughout the whole pulmonary tissue.

The next slide, this is from the right lower lobe. I am merely showing you the different sections from different portions of the lobes of the lung so that you might have opportunity to compare what we see in the actual tissue with the changes visualized in the Roentgenogram.

Again, this does not differ much from the sections of other lobes, focal deposition, linear deposition following the vascular tracts, evidence of anatomical forms of at least coarse emphysema.

The next slide. Now, if we examine this lung microscopically, we find that many of the vascular trunks are surrounded by a small amount of connective tissue which is readily seen in sections named specifically for connective tissue. Here we find no evident deposition of dust around this vascular trunk. As you perhaps know, following the deposition of dust in the lung, that dust is taken up by

phagocytes; it is mobilized. Those phagocytes, laden with dust, find their way ultimately, that some of those phagocytes, ultimately into the lymphatics about the vascular trunks, but this is a normal vascular trunk seen in the section of this lung.

The next slide. And then, we examine other vascular trunks and we find that in some instances, there is deposition of this black pigment, and there is a slight, very slight increase in the collagen that has been formed. You can see by this connecting tissue.

The next slide. And if we examine the lymph node of this man who, as you will recall, was fifty years of age, and worked for thirty-eight years as a motorman in a bituminous mine, we find dust which has been drained from the lung to the tracheobronchial lymph node, yet, there is no evident significant response to that dust which has become deposited in the node.

We thus - this case demonstrates that long occupation in a coal mine as a motorman does not demand the presence of large amounts of dust in the lung nor does that occupation demand the presence of significant pulmonary changes due to the inhalation of dust.

The next case, some cases exhibit changes as seen in this case, as seen cases with classical silicosis, complicated also by tuberculosis. This is the case, the chest

Reontgenogram of a fifty-year old colored man; occupational history, worked underground for twenty-eight years as a coal loader for eleven years, as a rock helper and rock helpman for thirteen years, as a truck helper for four years.

My X-ray on the right side, we see scattered throughout the entire lung field, areas of increased density, and there is also a fan-shaped area of consolidation, as you see in the upper third of the lung, within the region; that region there is also an area of relative rarification. On the left side, there are scattered throughout, small areas of increased density.

The next slide. The section of this lung, this happens to be the right upper lobe, exhibits scattered nodular lesions throughout all of the lung tissue. They are quite well defined, but yet not as defined as is the case with the classical nodular silicosis, in that the borders of these lesions are irregular, and if we examine many of them, we find that they are scattered in shape, with finger-like projections into the adjoining alveolar walls.

The next slide. And, if we examine many of these nodular lesions, we find that they demonstrate the deposition of collagen, arranged in typical fashion, which is classical for the reaction to free crystalline silica. In addition, we also find that the periphery of these nodules, is usually surrounded by a thick or thin collar, depending

upon the circumstances of exposure, of deep pigment, of black pigment. Furthermore, this pigment, and its associated slight reaction, deposition of collagen, extends into the adjacent alveolar walls. Examination of other lesions, we could demonstrate fibrosis, actual fibrosis, of hazy fibrosis in this case, of this ^{hyalinized} fibrous tissue, which is similar to the change of scene in silicotics, complicated by tuberculosis.

Thus, under certain conditions in coal mining operations, or certain coal mining operations are associated with the dissemination of dust, which, on inhalation into the lung, produce silicotic reaction, and some such reactions are also complicated by tuberculosis, as is present in this case. This complication is more evident in the next case.

Next slide. The gross section of this, the lung of a driller in a bituminous mine. He was a driller for nine years, been exposed to high levels of free crystalline silica. Now, we see, first the nodular lesions, scattered throughout the lung. We see that they are studded in shape. We see many of them on close inspection, that they project into the adjoining alveolar walls. We see the evident emphysema which is at best and coarse in type. We see large lesions and we also see coalescence of lesions which has given rise to a large massive shadow in the Roentgenogram, and close inspection shows obvious tuberculous complication.

The next slide also manifests the tuberculous complication such as here, coalescence of lesions with a massive area of fibrosis, complicated by tuberculosis with the production of an excavation in which numerous tubercle bacilla were found.

Now, the lungs of some coal miners show also the complicated features which accompany other pneumoconioses. For example, the next case, that of a white male subject fifty-three years of age, who had been employed for many years in a coal mine, a bituminous coal mine. Now, I can not give you all the details of his occupational history, because this is a relatively recent case and we are still seeking them.

The medical history, the patient developed a spontaneous pneumothorax, and we can see it on this side. Here is the edge of the lung, along the lung after an attack of coughing following a cold. The X-ray at this time shows a spontaneous pneumothorax on the right, estimated degree of collapse about sixty percent. The impression is given that within the collapsed portion of this lung, there is definite nodulation. On the left, several areas of ^{rarefaction} rarification are seen in the upper half of this line, with the loss of pulmonary markers which are usually present in a healthy lung. The impression is also gained here that nodulation is present.

The next slide, eight days later, now, a hydropneumothorax on the right has developed, a rubber catheter is inserted; there is a fluid line, and the area of rarification still exists on the left. The impression is gained that nodulation is present.

The next slide is a section from the right lower lobe in the area of consolidation seen Roentgenographically. It is a common fluid hypostatic pneumonic process. We will not spend much time on this particular feature of this case, but note also the scattered areas of focal pigmentation, and also the rather coarse emphysema.

The next slide, the right upper lobe section and also sections from the left lung, we see the isolated lesions which are not too numerous and here and here, stellar in shape, projecting along the alveolar walls. We see the emphysema, but what is more troublesome, in some of these cases of coal miners, and indeed it is not specific for the coal miner, we also see it in those individuals who have inhaled free crystalline silica, mixed with other components such as the iron miner, the magnetite miner. We see large areas of dust pigmentation presumably due to coalescence of the pigmentation, but we find areas of liquifaction necrosis within those lesions.

Now, this liquifaction necrosis is also very troublesome, although we believe that in the majority of

instances where coalescence of lesions has occurred where large massive areas of shadows develop in the test Roentgen, that that coalescence is due to a complicated tuberculous infection. Yet, there are those cases, with necrosis, with liquifaction and indeed with excavation, where we have been unable, with all means at our command, to detect the presence of the tubercle bacillus or to see evidence of reaction which characterizes tuberculosis.

Lest we're led to believe that coalescence and necrosis and liquifaction may be due to agents or to factors other than infection, and we have advanced the view that perhaps high local concentrations of dust may be responsible for some of the necroses, or that the necrosis seen in these lungs were - with mixed exposure, where there is present carbonization material in large amounts, where there is present iron oxide in large amounts, that perhaps this necrosis is a specific action for compounds in the dust, other than the free crystalline silica. Is it possible that the carbonation component, that the iron component, may have a power of absorption or adsorption and so keep localized the toxic substances, perhaps liberated from the free crystalline silica particles and some dust irritating particles which certainly may produce this necrosis. Certainly infection is considered - and I mention that - infection other than may produce tuberculosis, is a possibility,

and I submit that the demonstration of tuberculosis in some of these cases, can not be accomplished.

The next slide, if we take a micro-photograph of these areas of liquifaction, some of the things which I have said become more clear, as for example, here is a micro-photograph of that area of liquifaction. A connective tissue state, we see the typical collagen, sustained with connective tissue, as we see, which characterizes the response to free crystalline silica. We also see the heavy black pigment which has been inhaled and deposited in this coal miner's lungs and we see the area of necrosis and liquifaction. There is no evidence, histological evidence of tuberculosis infection. Then what is this due to? And I again submit the reasons for possible necrosis and liquifaction other than infection.

The next slide, and this is the character of the stellate lesion as we like to identify it, the irregular lesion which is not discreet, which is not the run or order of classical nodular silicosis or the reaction of the lung to pure free crystalline silica. We have here the whirling of fibrous tissue which characterizes the reaction to free crystalline silica and we have had the color of pigmented -- that collar of pigmented tissue, about the lesion which extends, as you see here, along the alveolar wall, and also the distortion of the adjacent alveolar spaces. This is an

important feature which I will call your attention to shortly.

The next slide, in addition to the area of liquifaction necrosis without detectable infection, is the focal reaction present about small deposits of coal dust in the lung. Now, this focal reaction has been described by Doctor Goffe. It has been described by others. It poses a problem which is difficult to solve and perhaps has not been solved. The focal deposition of dust with its reaction, the focal emphysema, is troublesome because it is a large factor in the respiratory disability perhaps of these coal miners, and here we have a case, a gross section of the lung of a coal miner, in a bituminous mine. He was a machine man and a loader for seven years. Here we see the focal deposition of dust scattered throughout the lung, different portions of the lung. The lesions are stellate in shape and you can readily see the distortion, the increase in size of the alveolar spaces, adjacent to these focal deposits. That then is the focal emphysema which has been so clearly defined by Doctor Goffe, and which has been referred to by Doctor Fletcher and his co-workers.

The next slide, and we stain these lesions specifically for a certain - with certain special stains and we note the heavy deposit of coal dust and we also note a proliferation of collagen, which permeates the lesion, and

we can see the irregularity, we can see the emphysema and also the projection into the adjoining alveolar space.

The next slide, another case -- no, this is the same slide, and here it is, showing you the infiltration of the adjacent alveolar walls with coal dust which has become deposited in the wall, the thickening of the wall, the relative or the degree of - the sparcity of the collagen, formation of collagen such as we would see if pure free crystalline silica were deposited in those walls and we see the large alveolar space, this is the anatomical form of emphysema as we see it.

The next slide. This emphysema is more pronounced and these, of course, are advanced cases and this - you can readily visualize, belongs to the - one of the stages so well described by Doctor Fletcher, of a bituminous miner for thirty-eight years. He was a mule driver for a number of years; he was a machine man and motorman; he was a coal loader for approximately twenty-four years, and he was a trackman.

His symptoms began approximately six years prior to death and they were increasing respiratory and cardiac difficulty. The X-ray, in addition to these areas of massive shadows, shows scattered focal shadows throughout the lung. There is also marked prominence of the left ventricular cells and the transfer diameter of the cardiac silhouette

is somewhat enlarged. The Roentgenographic interpretation was that in the presence of an adequate history of exposure to free crystalline silica, the changes visualized in the lungs are compatible with a Roentgenographic diagnosis of silicosis with conglomeration. Post mortem showed advanced arterial sclerosis, coronary involvement, cardiac hypertrophy and dilation.

The next slide. The gross lung in this case is heavily pigmented as you see here with black pigment and, of course, one can also readily recognize the coarse emphysema present. The next slide, and the section of the right upper lobe, the emphysema certainly is now most pronounced and most emphatic, and now again, we see the focal irregular deposits of pigment extending into the adjacent alveolar spaces, the small portions of the lobules of the lung are emphasized about these, and if we pick many of them up, there are large or more coarse emphysematous areas. There is, near the hilar region, this area of massive pigmentation. The tracheobronchial lymph nodes are enlarged.

The next slide. That was from the right upper lobe. This is the left lower lobe. Again, the emphysema now is more pronounced. Of course, gross emphysema, and the massive area of pigmentation. Of course, this is now the emphysema beyond the stage which is so troublesome. Of course, this is the progression, no doubt of this small

focal area, areas of emphysema which occur about the deposits of dust.

And now, the question arises, did the emphysema exist before the pulmonary deposition of dust? Would the emphysema have developed without deposition of dust or is the emphysema related to or caused by the deposition of dust in the lung? I am unable to answer the first two questions, namely, did emphysema exist before the pulmonary deposition of dust in this case, or would the emphysema have developed in these cases without deposition of dust? That demands a number of studies which I'm sure you will hear more about in subsequent days, but the question is, is the emphysema related or caused by the deposition of dust?

This picture of focal emphysema has been adequately described by Goffe and I should like to show some of his preparations which he has so kindly given to me.

This is the section from apex to base of a coal miner, age twenty, who worked on the coal face in a semi-bituminous mine. You will note that there are focal collections of dust in the lung and many of them. Some have coalesced to form larger collections. However, the point to be made is that there is little or no focal emphysema, associated with these deposits in this instance. Thus, the - not all cases showing focal deposition of coal pigment in the dust, in the lung, are associated with this

focal emphysema, as seen when the specimen is met. That does not mean that it may not develop, but certainly a case in point, is that not all cases of dust are accompanied by focal emphysema.

Another section by Doctor Goffe shows the deposits of coal dust in the lung and now the focal emphysema, associated with every - practically every deposit of this dust in the lung.

The next slide. Four points of reference at this time, I should like - I present the normal lung, a section prepared by us in accordance with the Goffe theory, and we see one lobe and another lobe of the lung of that side, and note the absence of pigment, the black foci that you see are cross-sections of asperducts; note the uniformity in size of the alveolar spaces. Note no focal emphysema or gross emphysema.

The next slide. Let us focus our interest now on this focal emphysema which characterizes the reaction associated with the deposition of coal dust in the lung and which is responsible, I believe, for most of the emphysema, focal emphysema, and thus responsible in large measure, for the respiratory difficulty experienced by some coal workers.

Here we see the deposits of coal dust in the lung. We see that the alveolar walls are pigmented with this deposit, that some of the alveolar walls are apparently

thickened and then ruptured with projections into the adjoining alveolar space.

The next slide. We'll go hurriedly now. The higher magnification shows the deposition of this coal pigment or coal dust in - again in both sides, sometimes about a vascular trunk, forming thick collars, but the point to be made here is that this pigment is not concentrated immediately about the wall of the vascular trunk, but it forms a thick wide collar, and as we look at it closely, we find also the deposition or formation of thin strands of collagen, showing that there is a reaction to this dust as deposited in the lung.

The next slide -- and a connective tissue stain of that lesion, again the vascular trunk, the - again the heavy deposit of black pigment which tends to obscure the collagen which is formed, and which makes histological study difficult, but note also the blue collagen which has responded to this deposition of coal dust.

Now, is the focal emphysema that we see associated with these focal deposits of dust, due to the collagen deposition and formation which we see here in these deposits of coal dust? Is it possible that these foci have reaction, that they act as anchors and so inhibit the natural function of the lung in these immediate areas, or is it because the changes -- the next slide -- which this merely

shows the area of higher concentration due to the response of free crystalline silica, due to pigmentation -- or is the emphysema due to changes in the vascular trunks? Or the blood supply to certain areas, as for example, here we see, a large vascular trunk sustained with elastic tissue? We see the lumen of this vascular trunk which is considerably narrower and smaller than normal. We see the elastic membrane and we see the deposit of coal dust about this vascular trunk.

The next slide. And, in other instances, again the wall of a vascular trunk, the inner elastic tissue and here we see the deposit of coal dust, and here we see the deposition of collagen, Actually involving and impinging upon these vascular trunks.

The next slide. Or, in other instances, we see the vascular trunk in the center of an area of pigmentation, almost occluded. Is it then possible that this focal emphysema which we see, and the respiratory difficulty in these coal miners, may be due to these changes in the vascular trunk?

The next slide. Or, is the emphysema and the respiratory difficulty, more directly due to the involvement of the pulmonary tissue per se? For example, in this instance, with deposition of this coal dust in the alveolar wall and its associated inflammatory response, could it be

due to that, and incidentally, we see here a body which is elongated with segments, and also with bulbous ends. They simulate the asbestos bodies which we see in asbestos workers. I have six cases in which we have discovered similar bodies. These have been reported by a good group, a large group, of other workers. Whether that asbestos body is due to really an asbestos body or whether it is a reaction to an asbestos fiber which has been inhaled incidentally, or an asbestos fiber from some fuses used, I am unable to say.

And, we see now, the stellate lesions, changes per se, extension along the collagen, extension along the alveolar walls, disportion of the alveolar walls, degeneration of the alveolar walls, loss of function of the alveolar walls and its accompanying distortion of the alveolar space, and emphysema. This slide again shows the thickening of the alveolar wall, obliteration of the architecture of that wall, loss of vascularity, the deposition of collagen.

The next slide -- and here is a section of the lung of a coal miner in which we see almost complete obliteration of the alveolar wall structures, replacement by deposition of collagen. The next slide -- and elastic tissue stains. We see adjacent walls of the elastic fibers quite prominent, we study the pigmented wall. It is difficult to see the elastic fibers, but I have not detected

any significant different in the character or amount of the elastic fibers which are present or absent in these areas of pigmentation.

The next slide. And we see here, complete obliteration of the architecture of the lung, by an involvement of the perimysium, pigmentation, hilization of the alveolar walls. The next slide -- And, is the emphysema and the respiratory difficulty due to obstruction of the major air spaces or passages? We do find areas of pigmentation, association of reaction, expansion of the alveolar wall, and repression of the small air space.

Next slide. In other instances, we find that the respiratory bronchiole is filled with mucous or with inflammatory obstruction. Is it possible then that the vascular changes, that the hilization of the alveolar walls, that the obstruction of the respiratory bronchiole, which we see in these cases, is responsible, as individual factors, for this emphysema and respiratory difficulty, or is any combination important?

Now, I should like to show you quickly a few other slides. The next slide. Although focal emphysema is discovered in the lungs of many coal workers, nevertheless, it is not unique for the deposition of coal dust in the lung. It accompanies also the pulmonary deposition of other types of dust inhaled by workers in other forms of

occupation, as for example, and quickly, the lung of a granite cutter.

Again, we see these areas of reaction to the dust, and we see emphysema about these foci. Here and here. The next slide -- A molder, in an industry, and we see the deposition of dust with its reaction, associated reaction, but not again the focal emphysema. The next slide -- I'm sorry, let's go back to that slide. This is a diathermaceous earth worker and we see the reaction, and the focal emphysema.

The next slide -- A molder, and we see the black pigmentation, focal emphysema localized about the pleura, about the areas of deposition of dust, and also the coarse emphysema. The next slide - a magnetite workers for thirty five years, again the focal emphysema and the areas of reaction to the free crystalline silica, which this worker has inhaled, with collars, irregular pigment extending into the alveolar walls, in this instance.

The next slide -- a hematite worker. Again, the emphysema which seems to be more prominent about the areas of dust deposition. The next slide, a graphite worker, again, the focal emphysema. The next slide - a carborundum worker; again, the emphysema which seems to be most prominent about the areas of deposition and about the areas of reaction. The next slide, a limestone worker, and again

the focal emphysema.

So I then submit that focal emphysema, although it may be more prominent in the coal workers' lungs, and although it may assume a greater degree of - may become more manifest in the coal miners' lung and may be responsible for much of the respiratory difficulty, particularly should it progress, yet it is not unique for the coal miners lung. Thank you. (Applause).

BY DOCTOR DRINKER:

Any questions to ask Doctor Vorwald? (No response).

I suggest we have a five-minute recess.

(Recess taken from 11:20 to 11:30 A.M.).

BY DOCTOR DRINKER:

The final paper this morning is by Doctor Fletcher on the Epidemiology of Coal Workers' Pneumoconiosis in Wales. Doctor Fletcher.

BY DOCTOR FLETCHER:

I'm afraid the title of this paper is a little bit wider than that. Strictly speaking, I'm going to talk about the Epidemiology in Great Britain, because some of the great interest in the epidemiological features of this disease in our country, is the difference in the prevalence of this disease in the different parts of the country.

So, first of all, for those of you who know as

BY DOCTOR FLETCHER:

I'm afraid the title of this paper is a little bit wider than that. Strictly speaking, I'm going to talk about the Epidemiology in Great Britain, because some of the great interest in the epidemiological features of this disease in our country, is the difference in the prevalence of this disease in the different parts of the country.

So, first of all, for those of you who know as

little about the geography of our country as I know about yours, I'm going to put a map up to show just where the coal fields are in Great Britain. We have, first, here, is the country.

The coal deposits in Great Britain are here in South Wales with little fields neighboring which are very unimportant in size. Then there is a very small field hidden at the moment, just coming out there, in Kent, some little fields in the Midlands, a very big field in Yorkshire and another one in Lancashire, a big one up in Durham, going out into the sea, another little one in Cumberland going out into the sea here, and other ones in Scotland.

And, where our work has been concentrated is in Wales, but we have also investigated the one up here in Lancashire and the one in Cumberland. By and large, the greater part of the coal in Great Britain here is bituminous, but in Wales -- next slide -- the coal grades are -- here is the coal field in detail, from anthracite over here through semi-bituminous or what we call steam coal out here, with bituminous coal out in the west. There is a complete spectrum of coals in South Wales from anthracite to bituminous.

Now, the disease of coal miners pneumoconiosis that I'm going to talk about was very fully described, I should

say pathologically described, over a hundred years ago in Scotland, and the disease then appears in British history to have disappeared. The experts said that there was no such thing now in about 1910, as the old miners asthma, the black lung of miners; it had disappeared and that was attributed to improved working conditions in the mines.

But then, as radiography began to be more widely applied in Great Britain, it was found that miners showed X-rays that looked very like silicosis and then the pathologists got interested again, and particularly in South Wales, described the sort of pathological picture that Doctor Vorwald has described to you, and in 1929, miners were admitted to Workmen's Compensation for the first time and some official figures as to the prevalence of this condition became available.

And here we start actually going through various legal delays, the first cases certified were in 1931. These are years along here (indicating on slide) and the height of the column represents number of men officially certified with the disease in each year from 1931 to 1947. There is a gap there because the legislation changed, and then there is '48, '49, '50. This is South Wales where about just over a hundred thousand miners work, and this is the whole rest of Great Britain where seven ty thousand -- six to seven hundred thousand, I should say, work; seven times

the number of men here, as in Wales. These cases after this gap here are not comparable strictly, because the legislation changed slightly and the standardx of diagnosis changed slightly but not very greatly.

You see the steady increase throughout the 1930's and then the very abrupt increase through the war. I'm not going into the reasons in detail; it was partly due to the increase of dust during the pre-war years, due to mechanization, also due to change of legislation during this time, and also due to the fact that during the war, miners could not get out of the mines except on medical grounds.

Wages went up in many industries and many men found pneumoconiosis a way to get lighter but equally arduous work, and so legislative, social, I think generally, epidemiological reasons for this tremendous increase. I just want to draw your attention to the increase of the size of the problem in South Wales.

These workers increasing during this time, over twenty thousand men were certified with this disease and removed from the industry. In 1945, over one in three of the unde4ground workers of two mines were removed from the industry for pneumoconiosis.

In great contrast to this tremendous prevalence in South Wales, by certified figures, we have the very much smaller prevalence in the rest of Great Birtain. The level

seems to be easing off in South Wales; it appears to be still rising in the rest of the country.

Well, now, in South Wales itself, there are also quite important changes in the certification prevalence as we look across the country. Over in the anthracite area here, this is a map in which little dots imply coal mines employing more than a hundred men, and these are contour drawings on mines with the same prevalence of certified disease over this period '40 to '45. The blacker they are, the worse they are.

The black here is over seventy per thousand per annum, and mostly the anthracite, with a tongue out here in the bituminous area, and the surrounding bituminous areas were very much lower prevalence.

Well, now, the Medical Research Council did a big investigation of this problem in 1938 and with Doctor Hart and Doctor Haslett and a lot of other people, and they confirmed, from survey work, this distribution of the disease in South Wales, the higher the rank of coal, the more the disease. They failed to show any direct relationship to any particular component of the coal such as the free silica or the ash. There was a general relationship to the total amount of dust. The disease, they pointed out, was quite distinct from classical silicosis and from their work, this rank of coal hypothesis, that high rank coals, anthracite

coals are more harmful than the bituminous coals, the soft coals, that came into being and has had wide currency, and that is Although, as I say, there was no direct evidence from their work, for this hypothesis because the anthracite mines were dustier than the steam coal, which, in turn, were dustier than the bituminous.

Well, now, to go on, owing to the very grievous situation in South Wales, the Medical Research Council set up a special unit in 1945, which I have had the pleasure and honor of being associated with since that time, and we have done a good deal of work into the epidemiological side - the epidemiological features of this disease.

The work has been done by my colleague, Doctor Cochran, and I really feel rather shy coming here and talking to you about work he has done. All that I am going to tell you about has been work for which he has been responsible and the credit goes to him and his team.

The objective of this epidemiological work has been, first, to try and see if we could get any explanation for these epidemiological differences as shown by certification figures, and secondly, to see if we could guide dust suppression by establishing safe limits of dustiness.

Now, the method -- next slide -- has been to study men and their dust exposure. Now, what sort of men

did we study? The sort of groups that have been used for epidemiological work have been the following: Miners attending hospital. I'm ashamed almost to put that on a slide, but I wouldn't do it except that quite prominent papers have been published on miners attending hospital, and clearly they are -- even a highly selected group, even if the doctor himself doesn't select his patients -- they have been selected for him on a very curious and unpredictable basis.

A lot of papers have been published on applicants or receipts for compensation. All the figures I have shown you so far as we have gone, are based on that sort of population, but again many-fold motives effect a man's application for compensation, and it's difficult to use that type of population for active work.

You then have more carefully selected populations. The ideal population would be all the men who have ever worked in an industry, whatever they're now doing, but clearly, that's impossible. What we can do is take selected cases, all the cases in a mine and selected occupations, all the underground workers, all the underground workers and surface workers, because miners tend to go from underground to surface from time to time, or complete mining communities. We have adopted, at various times, the populations three to six.

Now, the next important thing is to insure that you get all the population you're after. Here is, just to emphasize this point, the prevalence of disease according to our categories, 0, 1, 2, 3, simple pneumoconiosis, massive fibrosis, to a hundred consecutive cases attending the certifying panel at Collier. You see the first two are not represented, miners and ex-miners admitted to our ward, high proportion have exited; ex-miners in a town, this distribution; surface workers, and the designed underground population, and you will see that these three all agree fairly well, but the certified cases and the hospital cases show a gross distortion of the distribution of disease, although all these cases come from the same geographical area.

These are the collierys we have studied which have been in South Wales. Some of the collierys already have been studied by the Medical Research Council in 1938. We have re-read the films in our classification and used their figures. We have also, particularly carefully, studied some steam coal mines, this group here in South Wales. Steam coal is our semi-bituminous, and an anthracite mine with very little disease in it. This is another steam coal mine, and two mines in England with rather low concentrations of dust, two mines in England in Lancashire and Cumberland with very high dust concentrations reputedly, from

all we could hear, but very low certified incidence.

We hoped there to get a real contrast between the conditions in bituminous and anthracite mines in South Wales and the general relationship between dustiness and rank outside South Wales, to get mines which were really dusty but had low rank coals, and those are the two.

Now, this slide is attributed to Doctor Cochran, the percentage of the underground-surface populations which he has managed to X-ray in each of those mines are given there, and the only two cases, in this mine here and here, in which he was not fully in charge of the survey, there were early surveys, is the proportion below ninety-eight percent.

The important aspect of that is shown in two figures -- next slide. At these two collierys where we didn't get ninety-eight percent of the population, you will see that the proportion varies with age. The statisticians found difficulty in the casually young, and in the equally old. The middle-age you get easier. If you don't get ninety-five percent you get seventy-five percent; both of these collierys you got over seventy-five percent of the population, but you've got a distorted sample. It is essential to get a hundred percent population.

The next slide -- and one colliery where he got a hundred percent, Doctor Cochran analyzed the amount of

disease in relation to pneumoconiosis, inactive and clinically significant tuberculosis, on the radiograph and the first two hundred men came along willingly for examination. The second two hundred came along after some argument, and the third two hundred had to be frog-marched in, and you will see that people with pneumoconiosis come forth readily, the people with tuberculosis lag behind, so if you only get sixty percent, you have missed quite a lot of your disease. You get adversely distorted samples in relation to disease, a problem which is quite often omitted in reference to this point, and which is commonly omitted in survey work.

Well, now, some results -- next slide. Here are just the crude prevalences of these twelve collierys I have shown you, just the sort of thing you get from X-rays of these populations. Here is an anthracite mine, prevalence Category 1, 2, 3: Massive Fibrosis, PMF is short for that, quite a big range of these prevalences; these two English collierys with the low prevalence and you will see that this does, by and large, conform what is shown by certification figures, that if we're interested in dust exposure, we can't use this crude prevalence, and the question is what dust exposure should we examine in relation to this disease.

This slide shows figures from all these populations in relation to age, years spent underground and years

spent on the coal face, all lumped together, the whole lot, and you will see that in relation to age, there is a tapering off after the age of forty-five; there is no increase in the prevalence of the disease. This is Category 1. You take underground work, there is a smaller tapering off; when you take miners on the coal face, then there is a steady increase with increasing years on the coal face, and it was that argument and various other arguments that led us to concentrate our attention in relation to dust exposure to the coal face worker and to dust at the coal face.

And, what we have done is this: For our relationship between dust and disease, technique of field surveys, we have the unit, an X-ray team and a dust team. The X-ray team takes the X-rays and takes industrial histories. The X-rays are read by the duplicate reading technique I spoke to you about this morning. The industrial histories are carefully screened and from it we select pure face workers. They are defined as men who, during the ten years before the survey, have worked on the coal face for more than one year and worked in no other dust occupation for more than one year. We limit ourselves to ten years because our dust exposure may occur today, but the disease is produced in the past. We have, therefore, to multiply by a factor produced in the dust history in order to make the dust computation relative to the dust we have observed, and we do not

think we can do that over a greater period than ten years, so we restrict ourselves to pure face workers over ten years.

The dust team goes to the coal faces, all the coal faces in the mine using the precipitator, and they take dust measurements in terms of particles, c. c. or more strictly speaking, and I apologize, per millimeter, twenty-five microns, and we have now adopted a technique whereby the dust is not taken at fixed positions on the coal face, but is taken at the working places of colliers selected on a random basis, so that the dust measurements do represent the true measure of dust exposure of those men.

If the man selected is an absentee, well, no dust is collected that day and naught is contributed to the average, because the man wasn't exposed to dust that day. We introduced that relationship to the actual exposure of the man as closely as we can.

Then, we take the history of the mine as closely as we can in terms of production, ventilation, all the rest of it, in order to see how relevant this sample here is to the past and introduce a multiplication factor where necessary, but at the same time, through the filters, we take the analysis and we get an index of total dust dose expressed in particles per c. c. times years, that is the

concentration of dust multiplied by the number of years the man has been exposed to it, and on the other side, by multiplying that by the proportion of free silica in the dust, we get a total of silica dust.

Now, then, this technique of relating dust dosage to amount of disease is a standard pharmacological one, but I want to pay tribute to my colleague, Mr. Roach, who has done all our dust measurements, for the fact that he pointed out this particular method of expression to use, Doctors, who ought to have known better. Previously we were just taking an average dust concentration times an average years of all the men, but ignoring the great differences in the distribution of period of exposure between the different mines. What Mr. Roach suggested, we should do, is to take for each man the amount of time he had spent on the coal face, multiply that by the number of years and get, for each man, a dust exposure in terms of particle years, then group the man according to different groups of dust exposure and see the percentage in those groups showing radiological change and this is the result in all the pure face workers at eight of those twelve British pits where we had the dust information. Four of them we didn't do dust samples. Quite a nice sort of steep curve with the only dip here.

Well, now, we were interested in the difference

between the English mines and the Welch mines, so let's see what happens when we separate them. The dip comes right out and we find beautiful smooth curve, and this curve is fitted by less squares to these figures here which is quite different from the English.

These six Welch pits are all mutually consistent statistically, and statistically quite different from the English here. Here is the dust dosage, and they do show a quite clear difference in the relationship of radiological abnormality and dust exposure.

Now, then, why this difference? Well, it's natural that we should say 'Hurrah'; it is obvious that this is silicosis, anthraco-silicosis; you've only got to look at the American-European literature to see that, and it is the responsibility of silica, and so we take these five mines, the five not six, and we look to see. The range in ash content, nothing much in it; the high content is in one of the English mines; total silica, nothing in it; free silica, this should be 0.4 -- I'm sorry that's a misprint -- and you see, again that the English mines fall in the same sort of range as the Welch Mines; calcium; albumen; and the only striking thing is the volatile matter. These Welch mines happen to have this range where both the English mines have the low volatile. We haven't yet managed to study low volatile Welch mines, so we don't know how

important this is, but it's the obvious point at the moment.

Well, now, let's just look at the free silica dosage against dust. That's in the six Welch pits, and I want you to remember that other slide, that beautiful steep curve, and the very visible sigmoid adopted from it, and it's very clear that the silica is not relating to the abnormalities as well as the total dust.

Well, now, what can this mean, this difference between the Welch and the English mines? I think that in looking for the silica difference, we're really barking up the wrong tree altogether, and for this reason: That in talking about active and inactive dusts the other day, I think I suggested to you, or perhaps I didn't, but it's in my written paper, that you might define an inactive dust as one in which there is a lot of dust in the lung in relation to the amount of reaction to it, but as an active dust is one in which there is relatively little dust in the lung in relation to the amount of reaction.

Cork - quite a little cork, produced fibrosis. In coal, in iron, you get a large amount of dust with relatively little fibrosis, but if this difference between the English and Welch mines, were between the bituminous and the anthracite and the steam coal mines, if that were due to differences in the fibrogenic properties of these dusts, we would expect, pathologically, to find a greater reaction

in relation to the amount of dust in the Welch mines than we find in the English mines. Now, hitherto, the amount of material we've got from English mines is relatively scanty, but we have quite a bit and we don't see any such difference. If you look at an English bituminous miner's lung, Professor Goffe will say, 'I can see no difference in this lung from a Welch mine - miner's lung, except there seems to be less dust in it'. The amount of reaction in relation to the amount of dust is the same, but there does seem to be less dust in it, and that at present is our thinking, that for some reason or other the bituminous coal dust either doesn't get into the lung so easily or having got in, is easy meat to the phagocytes and they just walk straight out with it again, a very difficult hypothesis to test.

We're trying to do that with animal experiments, but it's going to be quite an elaborate statistical problem to get proof of that hypothesis, but that's where we're sitting at the moment. We think there must be some difference of some kind in the retainability of these dusts. I can say straightaway that it doesn't lie in the size distribution of dusts.

Well, now, that is as far as we have got in relation to these epidemiological differences. There is this difficulty. We can't yet explain it, and at the time, we

have set for the future, a plan to carry out simultaneous dust and radiological surveys at twenty mines scattered throughout the whole of Great Britain, which the coal board are organizing in collaboration with us, and we hope to overcome the inaccuracies of the historical factor in this way, and from follow-up surveys of this kind, to get really good evidence, more precise than we've got at the moment which, associated with and in addition to, animal experiments, may help us to solve the why's and wherefore's in this difference we have found.

Now, what about safe limits? Next slide. Here we have, for the Welch pits, where our working data is much more complete, these dose response curves for Category 1, and for Category 2, and more and Category 3, and here we have the dust dose. These are sigmoid curves, and are, of course, sound distribution curves. That is to say this curve here represents an ordinary - an ordinary normal distribution curve, and the fifty percent point is, of course, the most accurate point to take the relevant point on it from, and I have mentioned in my paper that our ratio between our categories is roughly speaking, but only roughly, linear, is about half as much dust again, at the fifty percent point here; two and three, you can see it isn't quite as accurate as it should be, but it's not bad, and this is our evidence that our categories are related to dust exposure.

Now, then when I putblished this in the Screed, I shall leave out these figures here, and I hope you will forget them, because obviously, this is quite a dangerous graph, because you can read up from here, if a man is exposed to five thousand particles per c. c., by the by, a thousand, approximately, particles is, I think I'm right, aren't I, Professor Drinker in saying it's approximately twenty-five to thirty million particles per cubic foot? I know you talk about these different things, but perhaps you better leave it untranslated at the moment.

Anyway, a man is translated to a thousand particles per c. c., which is about the official approved levels in Great Britain, for five years, fifty percent of the men will have Category 1 in five years. Ten years, fifty percent will have Category 2. Does it matter? I think we'll leave that discussion until Doctor Hugh-Jones has told you something about the disability.

Clearly, from this sort of chart, you can read off what will happen to men if they're exposed to these particular conditions for various periods of time, and obviously, I suppose to some of us, should forget the political consequences and just publish his results and let the workers and the employers fight it out, but I think that it's better at the present, to publish that graph without the figures on it for the future, so I hope you'll try to

understand why I omit the figures from the publication. It's also a matter of discretion, because in these figures we have got our historical factor which is a little bit of guess work, and these figures here are only relevant to the particular range of dust concentrations that we have observed.

We have found, for instance, that if you separately analyze years and particles, that for the same -- sorry, that for -- yes, the same period of time, twice the exposure produces more than twice the amount of disease. That is to say concentration is more important than time and these figures just represent an even weight of concentration and time. So much about safe conditions; that is our approach; I recommend it to you as a very valuable method of expressing this relationship.

I now want to turn to discuss what we refer to, conversationally, as the two-disease hypothesis which Doctor Vorwald has already raised with you. We find, in simple pneumoconiosis, we call it, these discreet capacities throughout the chest, and we find these massive lesions. Are the massive lesions just due to large quantity of dust in the lung or are they due to the action of some other agent and dust?

Doctor Gorfe's view is that these lesions are tuberculous, as Doctor Vorwald said. He finds tubercle

bacilla in fifty percent of them at post mortem, but in the other sixty percent he doesn't find them. He says that the histology looks to him like tuberculosis or the same. Doctor Vorwald won't support him there, and I can't enter into that, but we have got this large number of cases without definite overt evidence of tuberculosis at death. The explanation on the tuberculous hypothesis is that the tuberculosis has died out leaving its scar behind it, but I don't like that very much, because we do see cases that progress rapidly right up to death, and even in those cases that have progressed right up to death, we have two in which we failed to find any definite evidence of tuberculosis. It is possible that the tubercle bacillus initiates some process which then becomes self-propagating. We know that the dust in the lung is mobile, even in these little foci, because a man with simple pneumoconiosis, when he gets bronchitis, will cough up dust in his sputum many years after he leaves the mine, and it may be that once an infection starts in the lung, the rest of the lung tends to move up in that focus and then starts a propagating process that is due to the dust. That is a possible way of explaining these progressing lesions after tubercle has died out.

But we still believe that there is a lot of evidence for the tuberculous process and certainly for there

being two different disease processes involved and the evidence we have, I want to just discuss with you now briefly.

First, the Europeans say that these massive lesions are due to the action of silica. If so, we would -- sorry, will you just skip the next four slides; they're just pictures that Doctor Vorwald has already covered on the pathology. I think I can skip that, so come on to the next table. I just put this in in case Doctor Vorwald hadn't covered it.

Here we come to cases that Professor King in London and Nagelschmidt have done analyses on, on the whole lung in relation to the pathological group, normal, slight simple, marked simple, early coalescent nodulation and massive fibrosis, and you will notice there is no difference in these groups in proportion to the amount of free silica anywhere in that table.

If these massive lesions were due to those cases with a lot of silica in them, then we ought to find a higher proportion of free silica in these massive cases, than in the simple cases and you will see nine, ten, eleven, so we don't think that it's due to silica.

Now, evidence then, for their being two processes in the progression of the disease. We have followed cases over various periods of time, using other people's previous X-rays in this group, and our own X-rays here, and in simple

pneumoconiosis, with dust exposure, without dust exposure, and with dust exposure, we had eighty-one cases Category 1 to three without dust exposure. None progressed; two hundred sixty cases with dust exposure and eighteen percent progressed, so in smaller interval, our own X-rays, dividing no dust exposure, minimal, engine drivers underground, and under control code, none progressed without dust exposure; 21. -- 2.1 with minimal and 7 percent with dust exposure over this short period. Those are published already, so that pneumoconiosis progresses as much only with dust exposure.

Now, you go on to massive fibrosis; there are two features, one the attack of massive fibrosis on simple pneumoconiosis, and secondly the progression of it once it starts. Here again, small groups of cases, on here, forty in each group, but with over five years dust exposure and no dust exposure. This represents the attack, the number of cases attacked by massive fibrosis in that period, rather more in the no dust exposure group than the dust exposure. There is not a significant difference.

When you come to progression again, no difference, significant difference in the amount of progression or the attack rate of massive fibrosis in relation to dust exposure, so in relation to dust exposure, the two types of appearance, radiological appearance, behave differently.

Well, now, the way in which we've tried to further our knowledge of this question is an ingenious and courageous plan put forward by Doctor Cochran. We can't produce simple pneumoconiosis in animals and we don't think, therefore, we can really get relevant information about the tuberculous nature of massive fibrosis from animal work. Well, then, he said we'll have to use men and he took a complete population of a mining valley, the Little Rhondda, which is the Rhondda Fach, he took the complete population, thirty thousand and decided he would X-ray them all and he would then discover all the cases of tuberculosis in that valley, open tuberculosis, with the help of the regional hospital board, he would get those cases into hospitals and have sputum control of the majority - the remainder, rather, and by one-two testing, see whether he could reduce the rate of tuberculous infection or infectivity in that valley.

He would then compare the attack rate of massive fibrosis in that valley with that pertaining in a neighboring valley where no special measures had been taken.

This shows his success in X-raying that valley. I do want to pay tribute to Doctor Cochran for this remarkable performance. These are age groups along here, women here, men here, and this is the proportion of the total population established by private census at the time of the

X-ray survey, and the ground is well over one hundred percent. Where it gets over sixty, where it's difficult to get some of the old men and old women out, the survey was done in winter which is a mistake, because it's difficult to get them out and convince them their X-rays have any relevance to the health of the community, so you just compare this with the business figures published in this country, where a careful census has been taken according to county, and considerably lower figures, and this interesting drop in the young, the men presumably too busy, and the women with too many children to be able to leave and come to the X-ray.

But anyway, that remarkable achievement there, I think, shows that he has succeeded in getting nearly all the cases of tuberculosis, except some of these here, on X-ray. Well, now, the results of his experiment won't be available, of course, for five or ten years. But meanwhile, there are some interesting points from the prevalence point of view, which are relevant to our problem. This shows the numbers actually X-rayed, miners, ex-miners and non-miners, the adult females and the school children. The infants in the school areas were not examined, twenty-one thousand more.

In the course of this survey, Doctor Cochran and his team have - his team of four, in six months, they had

no less than twenty-thousand personal home visits to poor people in the valley.

This rather depressing picture shows the prevalence of pneumoconiosis in the ex-miners and miners in the valley. It shows there is quite a lot of pneumoconiosis, study of simple pneumoconiosis here without fibrosis in which the study of the attack rate and also quite a lot of massive fibrosis to study too, enormous population there, and the total numbers in this whole group are about nine thousand.

Now, this is quite a complicated one. I do apologize. The first point is that there is a logarithmic scale, and here I considered cases of infectious tuberculosis, let's say, with a positive smear on culture, cases of inactive tuberculosis, diagnosed on the radiograph as held inactive tuberculosis, and cases diagnosed as clinically active tuberculosis, let's say, cases requiring supervision, or in the cases where there is pneumoconiosis or massive fibrosis, and we found none in Category 1 in which the shadow looked like pneumoconiosis, but in many cases, on the others, it was awfully difficult to call the thing tuberculosis or massive fibrosis, so we put them here.

We have in category, infectious cases less than one percent, inactive tuberculosis and clinically significant cases. Coming into Category 1, there is practically

no infectious tuberculosis, and indeed in a survey of these, all these men and the neighboring valley, this one man is the only case out of four thousand miners in Category 1 pneumoconiosis, we had had a positive sputum on. There is decrease, insignificant decrease in the amount of clinically significant, but a rise in the prevalence of inactive.

Might we suppose that a little bit of coal dust in the lung increases the fibrogenic action of tuberculosis so that there is a tendency for a healed active scar and a reduction in the tendency for open tuberculosis, might that explain the facts? We then get a bit more coal dust in the lung, Category 2. Fibrosis is further stimulated. Massive fibrosis begins to appear. The inactive cases drop off as they're going slowly over there, but now infectious cases begin to appear when we get up to Category 3, when it goes right up to sixty percent.

Thus, in the case of massive fibrosis, of course, the Category is read on the background and your - it means unreadable, where it's that you simply can't read simple pneumoconiosis in the background. What's interesting is this reappearance of infectious cases and quite a portion or proportion of about one percent of the cases of massive fibrosis do develop positive sputum so that even though they are definitely these highly fibrotic lesions on our tubercular hypothesis, some of them may, for some reason or other,

and it's almost entirely in the age group over forty-five, break down and develop active tuberculosis, spreading tuberculosis.

It's perhaps of some interest that it's at that age that the mortality of tuberculosis rises in the general population. Perhaps it's something to do with resistance.

But, now, I can't speculate any further. Next slide -- and I must just point to the future for what is going to happen in this valley. And this is a map of the valley as it was in the first of September, 1950, with all the positive cases marked by pins on the map here. There is all the cases, in or out of hospitals. This is the same date, with the cases who were in hospital removed, and I think that it expresses the inadequacy of our present tuberculosis services, that is the very small impression that they make on the amount of infectious tuberculosis in a mining community, but the Regional Hospital Board said for this special purpose, they would open particular wards to which these men could be admitted and that was the position the 1st of October, 1951, the 31st of October, 1951, a year after the survey started.

This is relatively small, only about thirty-nine out of the original one hundred twenty-two are still in the valley, four months later only thirty-eight. The others are still in hospital or in their graves and the remainder

here are being carefully visited every month by a health advisor to insure they are really looking after their sputum, not going out to the pubs, and generally behaving themselves.

And what will be fascinating in the future will be to see what effect this has on the none too positive rate in the children, and then what effect it may have on the attack rate in massive fibrosis in those who already have simple pneumoconiosis in that valley, when compared with the neighboring valley in which this special procedure has not been followed, and I hope perhaps at your next Symposium, Doctor Cochran may be able to come himself, Doctor Vorwald, to tell you what he has found.

Well, now, gentlemen, I have spoken chiefly, on - about methods, just - and there is just time to refer to this chart. This is - I just have this one. It's a very provisional chart, this is the first of one year, but it's the mortality in age groups during the first year after the survey according to X-ray category and here is massive fibrosis, simple pneumoconiosis, Category 1 and 2, and that is the figure for the whole of England and Wales for the year.

Simple pneumoconiosis is just lined up completely with the normal population, normal male population; massive fibrosis is up, but I don't want to lay too much emphasis

on this. It's only one year and this particular kick here, which looks so impressive was due to four cases dying and two of them had carcinoma of the lung, so I think there is very possibly a significant difference up there.

But our point, I chiefly have spoken to you about methods and I'm afraid I have given you very few answers. I have, I hope, perhaps convinced you that there is some subtle difference between, at any rate, Welch and - Welch anthracite and steam coal dust and English bituminous dust. No same safe level would be applicable to these two mines.

I have touched on this two-disease hypothesis which we regard as very interesting and which we can not solve our problems at the moment, but we believe it to be perhaps more important than the problem of safe dust conditions and of simple pneumoconiosis. Doctor Hugh-Jones will show you this afternoon that disability in this disease is very largely attributable to massive fibrosis. We have some preliminary evidence that mortality is related to massive fibrosis. If simple pneumoconiosis is really not much more than an abnormal radiograph, then it's simple pneumoconiosis, that's caused by dust and if it is true that massive fibrosis is due to an additional factor, perhaps tuberculosis, maybe that we oughtn't to worry so much about dust concentrations which cause the simple pneumoconiosis, because the abnormal radiographs with little

disability and little mortality and what really we ought to do in our country is to remove tuberculous infection from the mining areas and if from the mining area, why not from the rest of the country.

I don't say that is necessarily the answer, but it's the way we're thinking at the moment. I think that it's going to be exceedingly difficult to lower our dust concentrations in British coal mines, the level of which no man will develop the radiographic abnormalities of simple pneumoconiosis. It may not be necessary to exert that close - that colossal an effort with all its economic consequences if we can protect the population from tuberculous infection, but first we've got to prove our point.

(Applause).

BY DOCTOR DRINKER:

Anyone wish to question Doctor Fletcher?

BY MR. URBAN:

I wonder if Doctor Abbott would like to comment - Doctor ^{Hammond} Hammond, would he like to comment on what he did along this line in Michigan?

BY DOCTOR DRINKER:

I couldn't hear that.

BY MR. URBAN:

Is Doctor Hammond here?

BY DOCTOR HAMMOND:

Well, I haven't been in contact with the iron mines for a long time, Doctor Urban, and I don't know that I have anything to add to what Doctor Fletcher said in that line, but I think it's - his experience parallels our own pretty well. As I listened to him, I thought that we were getting right back to what we here in Saranac Lake were taught a good many years ago -- when I say a good many years ago, I mean ten or fifteen -- that tuberculosis was the important thing, perhaps in these massive fibrotic lesions. We felt up there that every shadow we saw in an X-ray at that time, in our own bailiwick there, would probably have to be interpreted in the light of being tuberculous.

Now, I think that we're probably getting away from that a little bit, but I still think that it is perhaps the important factor, and I know that up there, we very definitely felt that if we could protect these people from tuberculosis, then we had a much better chance of getting disability from silicosis in that field. We had a situation there which was perhaps somewhat unique in this country, as far as the problems of prevalence of tuberculosis were concerned. We had some family histories that were, well, to say the least, they were unusual, but by intermarriage, younger people become diseased with tuberculosis, the parents are then going into the mines and contracting

tuberculosis at an early date, sometimes dying with their boots on. Those days now seem to have disappeared pretty well since we have better methods of exhaust ventilation in the mines, but those of us who worked in that area, and there are a number here, I think were pretty well convinced that in the majority of cases if we could control that, we could prevent disability from these cases.

BY DOCTOR MOTLEY:

I'd like to ask Doctor Fletcher, if in the epidemiological study they have made, any correlation with the study of the man? We have occasions to study buddies who were in the war together, in some cases even brothers, and one man may become severely disabled, where one man may show only slight disability. The factors we have thought of is sinus infection, mouth breathing. I know Doctor Schepper, when he visited my laboratory from South Africa, made much of the physical examination they gave the white men there before they permitted them to work in the gold mines. They gave them a very rigid physical examination and in correlating dust count, it seems to me there might be some correlation with the rapidity with which they develop changes, and it's a physical finding and I wondered if they had made any attempt to correlate that.

BY DOCTOR FLETCHER:

One man being taken and the other left. It's like

the Bible call, the last Judgment. We have got that too; one has massive fibrosis and the other hasn't. Two men work the same place, one man gets a huge mass of shadow and the other doesn't, one is disabled and the other isn't. You could say one has tuberculosis and the other hasn't. Whether in two men exposed to the same dust conditions, one will develop radiological abnormality and the other will not is a problem we discuss at great length.

We have, at the moment, got a group of miners who have worked on dusty coal faces for more than twenty-five years, in whom we can find no radiological abnormality, we hope to trace those up to see what happened and we hope to chase them into Professor Goffe's department to see if our X-ray is wrong or whether they can cope with disease so efficiently they don't take it, and I personally believe that some men have such competent phagocytes or something of that sort that they cope with dust that many others will be susceptible to.

My colleague works with animals, and he says that one will be exposed to dust and will cope with it, and the others may be susceptible, and he says that animals and men are the same in regard to dust. He may be wrong and I'm not.

In regard to South African experience, examination, prophylactics, Doctor Cochran has done a great deal

of investigation as to radiological activity and body type. By evidence, he has been able to sort his men into tall, thin, short, squat, and so forth, and there is no relation at all between those anthropological measurements and pneumoconiosis, simple pneumoconiosis; on the other hand, there is a relation between the tall-thin type and massive fibrosis. Either the tall, thin type are more prevalent to get massive fibrosis and tuberculosis, or when a man gets massive fibrosis and tuberculosis, he becomes thin and tall.

BY DOCTOR DRINKER:

Doctor Sander?

BY DOCTOR SANDER:

I'd like to ask, do you have a correlation, or have you made a correlation with emphysema in the various categories of simple pneumoconiosis, clinically significant emphysema, radiologically and by lung function studies?

BY DOCTOR FLETCHER:

As far as lung function studies are concerned, I'll leave that to Doctor Hugh-Jones. As far as radiological emphysema, I'm afraid we've been so far disappointed to read emphysema repeatedly on X-rays, that we have decided that a radiological reading of emphysema is worth about as much as the ink, the ink you bother to write it down with. But we do know our radiological categories are

unrelated to professor Goffe's focal emphysema. All we can say in the very advanced focal emphysema, Professor Goffe has got two or three cases, one of which published in his paper in the Faculty Radiologist, where there is gross focal emphysema, and the radiograph does show a kind of honey-comb pattern, so when we see a marked honey-comb pattern in a miner, we think perhaps he has got focal emphysema, and we have got about three X-rays of that kind, and the man has either died or we haven't got them. So I don't think I can say that; all I can say is where massive fibrosis develops in the last stage, there you can see the big ^{bulky} bully and you can be pretty confident.

BY DOCTOR SANDER:

Did you feel that your first film on the left, Category 1 has emphysema?

BY DOCTOR FLETCHER:

I feel it has, but I'm not sure, I'm not definitely positive.

BY DOCTOR SANDER:

You feel, logically, there is emphysema?

BY DOCTOR FLETCHER:

I feel there is, but if you haven't done any careful repeat reading of X-rays to classify them as emphysema and correlate them with the physiological findings, but I would only say that where the emphysema is gross, and

I think that one of these films fell into that type or category, then I would be jolly cross with the physiologist if he didn't find some evidence.

BY DOCTOR MAYER:

Now, we can discard completely the term anthraco-silicosis here, and to consider this as a form of anthraco-sis with infection. If so, it's going to effect our attitude toward compensation because our laws in New York State compensate the silicotic. Now, would you have us discard that term anthraco-silicosis?

BY DOCTOR FLETCHER:

Well, if it's going to deprive coal miners of compensation, I would say you must go on calling it silicosis, but I hope you'll be putting inverted commas on each side of the word, because I just don't think there is any evidence that the very small silica content, you may have noticed how very small the silica content of this coal dust is, I don't think there is any evidence that this silica content is responsible for this silicosis, even when Doctor Vorwald points to the diagram and says, here we have the characteristic response to silica, in the middle of the thing. Well, I am reminded that Doctor Hebbleston maintains that reaction to coal dust, with age, collagen may develop and it's common for any scar, so I'm told by the pathologists, for collagen to develop, and it may be a

reaction to aging as much as a reaction to silica, and I just simply say that the anthraco-silicosis implies to me that this is caused by silica, and I don't know anything in the world literature to support that hypothesis.

7m7 ->
BY DOCTOR McCORMICK:

I would like to ask Doctor Fletcher whether he would care to comment about the range of dust concentrations that you may find in British mines, and where he has been able to set up any sort of defense marks below which his early pneumoconiosis does not occur.

BY DOCTOR FLETCHER:

The range is very great indeed, and due to a variety of reasons, the anthracite mines that have been very dusty in the past for various reasons. First of all, in anthracite mines, there is no danger of coal dust explosions. Therefore, ventilation is not quite such an urgent safety measure, and in general, anthracite mines have been characteristically, have had sluggish ventilation in the past. There is much more short-firing anthracite mines and they have been very dusty. Bituminous mines have to be very vigorously ventilated because of the danger of coal gas and coal dust explosions, and by and large, the dust production from the bituminous coal doesn't seem to be so great.

At the moment, in Great Britain, there is an official standard of 650 particles for anthracite, 850

particles per cubic millimeter as approved conditions. Nobody states how long those approved conditions have got to be prevailing. That is to say, if a coal face is approved, if the dust concentrations rise for five minutes above that level or the average for a week or what. We feel that the right figure is the average for a month, and Doctor Richter is busy designing, has already designed a dust sampling instrument which will give an integrated sample for a period of a week and just give you one dust count to do at the end of a period of a week and we think that will be a sound basis.

Well, now, on those figures I pointed to you on the board, men will develop simple pneumoconiosis under those approved conditions. There is no doubt about that, so that the level will have to be lower if our objective is going to be to prevent all radiological abnormality, but massive fibrosis doesn't develop until a man has got at least category 2 in our experience, so that perhaps that's the level we ought to aim at, and we may not have to drop this too low to achieve that. Certainly in bituminous mines, we may have to be more strict than anthracite and steam coal mines.

There is one other point. I showed you a fall in the amount of infectious tuberculosis and clinically significant tuberculosis with a rise in inactive tuberculosis

in Category 1. Maybe we ought to give all our coal miners clinically active pneumoconiosis to protect them from tuberculosis. It is interesting that mortality figures for Great Britain, and I think other countries too, Great Britain quite definitely, have shown a low tuberculous mortality for coal miners.

Now, in Great Britain as a whole, Category 1 pneumoconiosis is very prevalent and there is very little 2 or 3. Maybe that is a - it's an advantage to a coal miner to have just a little dust in design, enough to protect him from tuberculosis, but not enough to disable him.

BY DOCTOR WARING:

I'd like to ask Doctor Fletcher if he has made any studies of the conversion of the ^{tuberculous test} tuberculous ties in relation to the development of pulmonary - progressive pulmonary fibrosis, massive fibrosis?

BY DOCTOR FLETCHER:

Unfortunately, we can't do that, because our miners have such a high disability rate. At the age of fourteen, when leaving school in Rhondda Valley where we had this figure, I think it's sixty percent mine coal, but the earliest, at the age of eighteen, which is the earliest we got a mining population, it's ninety-five percent, so our chance of watching that are very slight. We have got two miners in Category 2 pneumoconiosis, positive, and we're watching

them like anything, but unfortunately, one got nephritis and died so we have only got one, but in the future in Rhondda Valley, we shall have opportunities for that sort of thing.

BY DOCTOR FRIEDMAN:

I'd like to comment on Doctor Fletcher's remark that we could detect some of the difference in the body in the people who have conglomerate lesions in the X-ray and the physical appearance. We're studying about two thousand soft coal miners in Alabama; we have failed to establish any correlation between body build and the appearance, whether it be a simple type of pneumoconiosis or whether it should be a fibrotic variety. However, we have observed that where there is a coalescent factor and the infection becomes overwhelming, in the event he dies in a state of malnutrition, it becomes evident.

Now, the second point Doctor McCormick raised about the dust levels. I feel, and I think it is true too, that regardless of the safe level of dust in which a man works -- and that is within reasonable limits of safety -- if he works long enough in that environment, he will obtain the same type of exposure, let's say, in twenty years in a safe environment that a man would obtain in an unsafe environment, in a lesser period of time.

I don't think we should place too much emphasis

over the long range period of time on safe levels of dust, because of the individual exposed long enough to dust, he will get the same effects over a long period of time which he can in a short period of time in higher concentration.

BY DOCTOR BRODKIN:

My name is Brodtkin and I was going to ask a simple question. The incidence of scars is frequent enough. Has anyone made any correlation between the tendency to keloid formation and the extent of fibrosis?

You take two individuals. Each has the same scar over an area; one will develop -- irrespective of any infectious bacteria, one will develop a large keloid, the other one will develop a fine hair-line scar. Has there been any correlation of that?

BY DOCTOR VORWALD:

Who is he asking that of?

BY DOCTOR DRINKER:

Were you asking that of anyone in particular?

BY DOCTOR BRODKIN:

Well, anyone.

BY DOCTOR VORWALD:

I have no evidence - I have no evidence to show that, for example, individuals or subjects from the faces who develop keloids very readily, that they respond more readily to the deposition of dust in the lungs with the

formation of collagen. I have no evidence, but we do know of course, that collagen does form in the Negroid race and the Caucasian, people from Japan and elsewhere. Perhaps Doctor Orenstein could tell us something about that. Doctor Orenstein, Doctor Orenstein, can you comment upon whether there is, in the Bantu, the colored individual, presumably a greater tendency to develop keloids, and if so whether he is more prone to develop collagen in the lung due to the deposition of dust, than is one from a race without or not prone to develop keloids.

BY DOCTOR ORENSTEIN:

Why, actually, it used to be one of the extraordinary museum specimens that we had and still have, of our Bantu lungs and spleens and livers with enormous things that you used to call tuberculoma, huge deposits, and nowadays you don't see it any more. The specimens are old. I don't know why, I have no explanation. It would be purposeless to detain you here with trying to explain differences between what you see in colliery workers in, say, Wales, to what you see in the workers in Johannesburg, where they work in a high percentage of silica, and historically, it's a very complicated thing, so I don't think you want to take up your time, because I can only say this, in the Bantu and in the white man, the type of pneumoconiosis which is so in the first two and a half decades of

this century and the type you have today are as different as anything can possibly be, pathologically, microscopically and clinically.

BY DOCTOR VORWALD:

Certainly we know from evidence there is a species difference, as for example, the white rat is much more prone to develop collagen in a shorter period of time than is the guinea pig or the rabbit, to the pulmonary deposition of free crystalline silica, but again, I certainly have no evidence that the colored race responds with more collagen under the same conditions of exposure than does an individual from the white race.

Now, perhaps Doctor Fletcher would like to comment. Fletch, Charles, do you want to say anything about that?

BY DOCTOR FLETCHER:

I have nothing to say about that; I would hate to be charged with designing experiments on this problem.

BY DOCTOR ABNER:

Abner, Buffalo, New York. Isn't, logically, a keloid the same as a fibrous tissue appearing in the lung following silica and if it is, should the response to X-ray therapy be the same.

BY DOCTOR VORWALD:

Well, histologically, the actual architecture of the keloid, histological architecture is different than the

architecture we see in the silicotic nodule, but basically it's collagen and I assume formed in a different way. I can't comment as to whether the collagen in the lung would be as amenable to change under the influences of X-ray therapy as might be the keloid.

BY DOCTOR DRINKER:

All right, Doctor Friedman.

BY DOCTOR FRIEDMAN:

Half of our population in the coal mines in Alabama is colored and the other half is white. We have not been able to detect any difference at the autopsy table or in the Roentgenogram on both. Now, on the other hand, we have an opportunity to examine one Negro who had massive keloids, but his bone specimen didn't show any inflection of what his skin showed in keloid formation. There was absolutely no correlation that we could detect in our study. You can find just as much fibrous tissue in the white man as you can in the colored man and I say, as Doctor Vorwald pointed out, there may be more of an individual difference than there is a difference between the races. We can't detect any racial different in response to the dust.

However, only one thing, we should say in conditioning this statement, is the fact that the colored population in our community live in a little less pleasant

environment. They don't have quite the economic facility that some of the white people have and the mortality rate in our colored miners that work underground is greater.

BY DOCTOR BOETJER:

I wanted to ask Doctor Fletcher if there is any difference in the prevalence rate in the general community in the English mining area which you were showing us, compared with the Welch mining area?

BY DOCTOR FLETCHER:

There is an interesting point there. The male population, mortality rate, is very specific, White Haven Borough Council and the Rhondda Urban District Council, here is distinct lines of pneumoconiosis, here female rates are almost identical and the children mortality rates are almost identical, but it is interesting that the male mortality rate in the Rhondda shows a deficiency, particularly in the age groups.

Now, if you add in to the age mortality, the male pneumoconiosis mortality which is nearly all pneumoconiosis, massive fibrosis, then there is another argument here for supposing that the men dying here of massive fibrosis would have died of tuberculosis if they hadn't had their massive fibrosis.

BY DOCTOR DRINKER:

Can you people in the back of the room hear this?

Can you hear the speakers all right, because I must say I can't hear when they ask me, but it may be a reflection on me. Can you hear the papers all right, you in the back of the room?

(General response indicating "Yes").

BY DOCTOR MILLER:

I wish Doctor Vorwald would comment on the idea that concentration of exposure was more important, the duration of exposure was more important than the concentration.

BY DOCTOR FLETCHER:

Excuse me, the other way around.

BY DOCTOR MILLER:

No, I believe Doctor Friedman said that the duration of exposure to a noxious dust was more important than the concentration, maybe I misunderstood.

BY DOCTOR FRIEDMAN:

I said that the duration of exposure has to be taken into consideration, it was also a very important factor.

BY DOCTOR MILLER:

Oh, not the sole factor?

BY DOCTOR FRIEDMAN:

No, sir.

BY DOCTOR FLETCHER:

I might just answer that point. The evidence we

have got on that point is simply this, that if you - if you do your time, your response curve here, the miners who have been exposed to more than a thousand particles per c. c. and less than a thousand particles per c. c., we have got time along here and percentage effected here, then the people in the high -- that's concentration -- show a curve like that; the people in the lower dust concentration a curve like that, so that just for equivalent particle years, you have a smaller response if the actual concentration of dust was level, and that is purely provisional, and is not of statistical significance there, but it is a trend that makes us doubt the applicability of our figures, necessarily, to the whole range of concentrations.

BY DOCTOR VORWALD:

I should like to make one comment and that is with respect to the free crystalline silica content of the lungs. Doctor Fletcher pointed that out and he used the low free crystalline silica content of the lung as a basis for saying that probably free crystalline silica is not a factor in the reaction of the lung to the deposits of coal dust, correct, Doctor Fletcher?

BY DOCTOR FLETCHER:

No, there is no more free silica in massive fibrosis than simple, so the massive fibrosis isn't due to more silica in the lungs in the fibrosis cases than in

simple pneumoconiosis.

BY DOCTOR VORWALD:

Well, some of our experience would be different than that, in that we have analyzed some of the massive fibrosis lesions and some without, and we have found higher free crystalline silica substance in the higher massive fibrosis lesions. I might emphasize though that there are those where we have not found that.

I would like to say also, with regard to the free crystalline silica in the lung, we have examined many lungs of normal individuals, of individuals who have been exposed to free crystalline silica, but without silicosis, and of individuals exposed to free crystalline silica with silicosis.

Now, there is a wide range of values observed in the normal lung versus the lungs of subjects without silicosis, but exposed to free crystalline silica, and the lungs of subjects with manifest silicosis. There is a wide range, and this range overlaps, so that except for the extremes, the two extremes, we believe that there is relatively little value or one can give a little index to the amount of free crystalline silica in the lung as related to the degree or character and extent of pathology within the lung. There is no correlation.

BY DOCTOR GREENBURG:

How about the time factor, Doctor Vorwald, did you take that into account?

BY DOCTOR VORWALD:

Well, I can't specifically give those figures; it's over a long period of time. In other words, here are fifteen normal lungs which we analyzed and they all have free crystalline silica, a certain range from a low to - I forget what it was - twelve percent, Mr. Durkan, do you remember those values?

BY MR. DURKAN:

No.

BY DOCTOR VORWALD:

Then we have sixty or seventy-five lungs of individuals who have been exposed to silica by reason of their employment, yet without silicosis pathologically, and they have a range of free crystalline silica value which is broad that way.

Then we have forty-five or fifty silicotics, established silicosis of industrial workers, and they do have a large range, but this range overlaps so that except for the extremes, we can not place any correlation between the amount of free crystalline silica detected in the lung by our methods and the degree and character, extent of pathology, silicotic fibrosis in the lung.

BY DOCTOR GREENBURG:

Well, that is another way of saying what has so often been said, namely, that there is a high degree of persons susceptibility in individual variation too?

BY DOCTOR VORWALD:

Yes, but maybe Doctor Pratt wishes to comment on that.

BY DOCTOR PRATT:

I think there is one important thing more that should be added to that ^{comment} content, that is that all that work was based on the percent of silica in the lung. We realize that the amount of silica in the lung ash correlates ^{poorly} fully with the amount of silica ^{is} in the lung, but very recently using a simple technique to determine the amount of silica in the ^{in total amount} lung, that correlates a great deal better with the amount in animals and a number of animal experiments show nice correlation, ^{between} but the amount of disease estimated in the sections and the amount of silica in the lung.

To quote one example of a case in which a lung showed fibrosis, another lung showed simple nodular silicosis. In each lung the percentage of silica was the same, but on the basis of total amount of silica, there was about one gram of silica in the whole lung, with silicosis, and one gram in the lung showing fibrosis, even though the percentage was the same.

Obviously, the difference is the ash from the

reaction dilutes the silica which is present, and I think Doctor Fletcher's figures could be a lot more convincing if they were based on the total amount of silica.

BY DOCTOR DRINKER:

I suggest we better adjourn and continue the discussion this afternoon. The next meeting is at 2:30 this afternoon.

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(Adjournment taken from 1:10 to 2:35 P. M.)

PNEUMOCONIOSIS IN COAL MINERS (Continued)

2:30 to 5:30 PM
Sept. 23, 1952.

Chairman: O. A. Sander, M. D.

Pneumoconiosis in Coal Miners in Alabama

Louis Friedman, M. D.

Discussion

Pulmonary Function Studies of Coal Miners in
Pennsylvania and West Virginia

Hurley L. Motley, M. D.

Pulmonary Function Studies of Coal Miners in
Wales

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BY DOCTOR SANDER:

Let's have the meeting come to order. We'll carry

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BY DOCTOR SANDER:

Let's have the meeting come to order. We'll carry

on with Black Tuesday, the Pneumoconiosis of Coal Miners day, and see if we can confuse and confound you a little more this afternoon. First of all, doctor Vorwald has an announcement.

BY DOCTOR VORWALD:

It's a very simple one, and that is we are requested again to remind you, those of you who wish to attend the banquet, to please register before tomorrow noon for the simple reason that the hotel manager must have the number of people who are coming so that he can prepare the adequate amount of food for the group and set up the number of tables that might be requested, so please register for the banquet and know also that the ladies accompanying you are invited to the banquet.

BY DOCTOR SANDER:

The first speaker this afternoon has had a large clinical experience with a group of bituminous miners in Alabama. He will tell you about it, his clinical experience with this group. Doctor Louis Friedman of Birmingham.

BY DOCTOR FRIEDMAN:

My remarks this afternoon will be based upon our experiences in the study of about three thousand soft coal miners in Alabama. When I was much younger and before I had the advice and counsel of people like Doctors Sander, Vorwald and Doctor Lanza and Doctor Hussey, I accidentally

BY DOCTOR FRIEDMAN:

My remarks this afternoon will be based upon our experiences in the study of about three thousand soft coal miners in Alabama. When I was much younger and before I had the advice and counsel of people like Doctors Sander, Vorwald and Doctor Lanza and Doctor Hussey, I accidentally

called my disease 'anthraco-silicosis', with some justification, I thought. They have subsequently persuaded me, energetically and in a very friendly manner, to convert the title of my paper to 'Pneumoconiosis in Soft Coal Miners' and then when I mentioned that to Doctor Lanza, he added 'In Alabama'. I had nothing to do with the title.

The pulmonary dust disease, as we see it in soft coal miners, is a disease which produces disability, both on a structural and functional basis. My remarks to you this afternoon will be from the point of view of a clinician as Doctor Sanders indicated, and our great concern is the correlation of X-ray findings, clinical history and functional studies with the total picture presented to us by the patient.

Unfortunately, it is not always possible to describe disability on the usual standards in the case of coal workers' pneumoconiosis. Only so frequently do we find a lack of Roentgenographic findings and nevertheless, we are convinced that the man's occupation had something to do with his present disability.

I would like to show you -- slide, please -- in the first slide, I would like to show you some of the terminology that we use in Alabama, at least we use in our clinic, to simplify the problem. Now, in presenting this classification of clinical disability and Roentgenographic

classification of the disease, I do not mean to take issue with Doctor Fletcher in the wonderful work that he has done in breaking this Roenggenographic picture down to its fine elements. To me, this is the clinician's idea of the classification in which you ascribe very little, in which you do not rely upon your X-ray for further than identification, and in some instances only do you use it on the basis of very pronounced structural changes, to determine the fact that this man must obviously be disabled. We merely use the Roentgenogram to classify the particular disease and call it pneumoconiosis of soft coal workers, and then we use our clinical studies and laboratory studies to determine whether there is any disability.

We consider, in sensing a man's disability, that a man is either disabled or not disabled as a result of his occupational disease, and on that basis, you can have a disease which is disabling, temporarily disabling, as I will show you later in the slides, or you can have a disease which is non-disabling, and for those of us who so frequently find it necessary to sit on a fence, we have the fourth classification of possibly disabling disease, until we can make up our mind three months later when you come back for another X-ray and examination.

From the point of view of assessing disability, I would like to take this moment to say it is very easy for

a doctor to rely upon his functions studies after they're done in the proper fashion and say 'This man has forty percent disability'. Well, that is from a medical point of view, but you take a coal miner who has no education and who has worked in a coal mine all of his life and at the age of forty-five, fifty or fifty-five, suddenly finds himself with a disease that will progress is he is exposed to his present occupation for any further time, to my way of thinking, and I think that is shared by some of the members here today, that man is actually a hundred percent disabled, because he can no longer find his economic place in society to gain a livelihood and take care of himself and his family.

Now, as regards the Roentgenographic classification, you will notice that we have Type 1, which is a linear variety, a linear change in the X-ray which probably corresponds to Doctor Fletcher's simple pneumoconiosis, and then the Type 2, the nodular variety which we do not see very frequently in coal miners, and Type 3, the mixed, one in which you see a picture of linear changes and nodular changes in the Roentgenogram, Type 4, the conglomerate coalescent variety, maybe a progressive change from Type 1, 2 or 3, so you may have conglomerate coalescent nodular pneumoconiosis of soft coal workers, and finally in Type 5, the Roentgenogram itself, you can see that this is a

complicated case, pneumonia, bronchiectasis, spontaneous pneumothorax or some other complication which we are able to discern from the Roentgenogram and, therefore, in sending a communication to another doctor, another clinician, who doesn't get too deeply into the breakdown of Roentgenographic types of disease, we could say that this man has non-disabling linear pneumoconiosis of soft coal workers and immediately he knows what we're talking about.

Next slide -- No, on this first slide, you'll see -- I wonder if we put the lights off whether we couldn't have a better view of the screen. Now, on this first slide you see the X-ray of a twenty-seven year old boy who died of acute monocytic leukemia, after working only three and a half years in a soft coal mine, and if you notice his Roentgenogram, there is nothing particularly revealing about it and it could be classified as increased bronchio-vascular markings.

However, the next slide -- you skipped -- Now, this slide shows a cross-section of his lung prepared by Doctor Goffe in Wales, and you will see that he already has the deposition of coal nodules throughout his entire lung. Now, I use the term coal macules to correspond with Doctor Hebbleston's description of the disease pathologically. Now, if you will notice, although he has these

marked macules throughout his entire lung field, there is no evidence of nodulation in the X-ray and we have no evidence from the X-ray that this man even had any changes compatible with an early type of pneumoconiosis. However, those of us who work with the disease and know that the changes, the early changes or the nature that we saw of the X-ray of the patient, would suspect that something was present in this man's pulmonary tissue that wasn't present in a normal individual's chest.

Now, this is an example of another linear variety of pulmonary dust disease in soft coal workers and, although this patient doesn't look particularly disabled from Roentgenogram, he is unable to participate in any gainful occupation after having worked thirty-seven years as a coal worker in Alabama.

Next slide. Now, this is a slide also showing only increased bronchovesicular markings and nothing particularly significant. This man died of acute lymphatic leukemia after having worked thirty-five years in a soft coal mine. I would like for you to look at the apices of this lung very carefully, because the whole lung section in this case will not correspond at all with what you have seen from this man's X-ray.

Here you see another whole lung section prepared by Doctor Goffe, and you see the focal emphysema, the coal

deposits and some evidence of silica spread throughout this man's lung field. Again, pointing out to us very clearly that you can not rely upon the Roentgenogram to determine either function or the degree of pathology which you will find when you examine the tissue at necropsy.

Now, this is an example of nodular type of pneumoconiosis in the soft coal worker. That's what you think and that's what I thought, but it isn't so. This man worked in the coal mine, but he actually wasn't a coal miner. He worked on a motor up a haulage way and for thirty-three years, he sanded a track in front of him and he was inhaling pure silica during that entire period of time and it's only in those instances in Alabama that we are able to find both a motorman and people who do a lot of drilling in hard rock, evidences of the nodular variety of pneumoconiosis in soft coal workers, so actually this man, although he worked in a coal mine really wasn't a coal miner and he actually has a true case of silicosis.

Now, this is an example of a seventy year old man and, as you see, he has both nodules and linear changes in both lung fields symmetrical, and you would think from this X-ray, that the man should have a great deal of disability. On the other hand, he never complained of any signs and symptoms referable to his - to his lungs, no dyspnea, no cough, and he died of a carcinoma of the stomach.

I want to apologize for the fact that this was not prepared in color, but as you see in this whole lung section interpreted by Doctor Goffe, indicates that this man not only had coal but a great deal of silica in his lung tissue. At this point, I would like to say that we have two groups of patients in our clinic. We have coal workers and ore miners and, in our experience, it appeared that the coal miners suffer greater disability from their exposure to coal dust than do people who work in ore mines.

In this particular case, you can see the extensive changes in this man's lung field without any evidence of clinical disability for a man of his age compared to another man who had to work in a coal mine of the same age. The next line is the microscopic section and you see here, you see the silica nodules, you see some of the silica deposited in whirls and in the middle of the nodule, you see the coal dust.

The next slide. Now, this is an example of a Negro who had worked twenty some years in a coal mine who came in complaining because of pain in his chest. We examined him very carefully and even though we maintain a high index of suspicion - of suspicion that this man may be suffering from pneumoconiosis of soft coal workers in Alabama, we found no evidence to substantiate our clinical impression that he may have the disease. Therefore, we did

some additional studies and in the esophageal examination with berium, revealed a little nick in his esophagus and because he had a widened mediastinum, we decided to explore this man and see what his trouble was.

Well, that surgery, Doctor Charles Donald, a thoracic surgeon, performed this operation, found that both of his lung fields were full of nodular masses which we were unable to see in a Roentgenogram, and this microscopic section was taken from the mediastinal lymph node which was the size of a tennis ball, and as you see in this mediastinal node, evidences of silica in whirls and coal deposits, so this was another example of more pathology being present in the pulmonary tissue than we were able to discern in the Roentgenogram.

Now, this is an example of a man who died in his seventies, and of a cerebrovascular accident. He suffered from hypertensive arterial cardiovascular disease, and if you notice his Roentgenogram, examine it carefully, you don't see anything too revealing. You see the evidence of increased markings and for most of us who are really interested in disease we could possibly read into this focal emphysema.

Now, notice the left lung and notice the right lung and notice the next slide that appears. Focal emphysema, evidence of the coal macule degenerating and causing

the focal emphysema and similarly, in the left lung over there, so you see again the evidence of the disease pathologically is much more extensive than you can discern in the Roentgenogram.

Now, this is only a clinical example of what we term conglomerate coalescent pneumoconiosis in soft coal workers. Here you see a solitary nodule in the right apex. Now, I would like to say this, in the ensuing slides, you will see more examples of conglomeration and coalescence and we experience a great deal of difficulty in attempting to prove the diagnosis of tuberculosis in these patients.

By very exhaustive laboratory studies which include smears, concentrates, cultures, guinea pigs, and all other varieties of tests which may ascertain the presence of tuberculosis - of tubercle bacillus, we only too frequently find that we are unable to demonstrate it. Perhaps Doctor Vorwald's contribution last year or the year before on the B. C. G. vaccine in silicotic animals could possibly explain the fact that we have evidences of infection or at least evidences of conglomeration, and coalescence in those places in the lung where you ordinarily find tuberculosis, and still we are unable to find a tubercle bacillus. Perhaps it doesn't take too many tubercle bacillae to set the process in motion here and later on, we just can't find them either in life or in death. As a matter of fact, during

life, we only find tuberculosis in our patients in about ten or fifteen percent of the cases, whereas we're able to double that at the necropsy table.

This is another example of conglomeration and coalescence in a soft coal miner, and again we were unable to prove the existence of tuberculosis, and incidentally, in this particular case, the man's tubercular test was negative. Recently, I have come to the conclusion, whether it's right or whether it's wrong, that whenever you have conglomeration and coalescence in assymetry in the lung fields of people who serve - who suffer from pneumoconiosis, I believe that we are justified in ascribing the assymetry of the conglomerate and coalescent lesion to a superimposed infection, more than likely tuberculosis.

Next slide. This, now, is an example of a fifty-five year old coal miner who has bi-lateral conglomeration and coalescence which probably fits the terminology of angel-wing distribution of the disease. This man had open hemoptysis - the man had hemoptysis on frequent occasions and we examined his sputum on the time that he had hemoptysis and the times he didn't have hemoptysis and we have still been unable to prove a tuberculous infection in this particular case. Of course, he's terribly disabled, not only structurally from what we see in the Roentgenogram, and what we know will happen to the man, but also on the

basis of functional studies.

In the next slide you will see what I presume to be an ensuing stage of this man's disease. Here you see cavitation in the right upper lobe and a similar type of lesion in the left lung as we demonstrated in the previous slide. Of course, we had no difficulty in this particular case in proving the existence of tuberculosis, tuberculosis in this patient.

Next slide. I believe that the slide before the one I just showed can go to either the tubercular stage that I just showed you or else it can develop the conglomerate coalescent features of the X-ray on the right or it may go on to develop the changes noticed here on the left, in the left slide, the left X-ray.

It may be of interest -- one moment, please -- it may be of interest to you to know that both of these patients at autopsy had tuberculosis and exhaustive studies in both instances over a period of several years, failed to reveal the presence of tubercle bacillae, and at the autopsy table, the tissue obtained was examined by Doctor Casey, the pathologist in our community. He had a great deal of difficulty in finally obtaining sufficient evidence to call this tuberculosis, but he did find the organism.

Next slide. Now, this is an example of another coal miner who worked twenty-nine years in a coal mine --

excuse me -- twenty-six years in a coal mine and three years in an ore mine. Notice the conglomerate and coalescent lesions in his apices. This is not an unusual picture in Alabama, where our coal miners have to do a lot of drilling, so we have a sufficient admixture of coal dust and silica to present this type of picture. I want you to notice the lower parts of both of these lung fields and the upper parts and then in the next slide, you can see the conglomerate coalescent lesion in the pathology and in the lower half of the lung field, you see nodules or macules of coal which were not discerned in the Roentgenogram, indicating again that the coal macule itself is not the radiopaque - has not the radiopaque character which we ascribe to silica or fibrous tissue, and if you were to rely completely on the Roentgenogram, you would miss so many cases of pneumoconiosis in soft coal workers.

Additionally, I want you to notice the focal emphysema in the lower half of this man's lung fields.

Now, this is an example of pneumoconiosis in a soft coal worker with cavitation in the right upper lobe. I was convinced, as well as everyone else, that saw this case, that this patient had tuberculous infection superimposed, but instead he turned out to have -- next slide -- what Doctor Bolliter has described as a septic necrosis. We were able to find the cavity, but we were never able to

find any tubercle bacillae. Again I want you to notice the extent of symple pneumoconiosis in the right lobe and again in this lung over here, notice the large amount of coal deposits and the cavity without any evidence of tubercle bacillae, both in life and in death at the necropsy table, and very careful studies.

Next slide. Now, the problem frequently comes up, and the question is often asked, is pneumoconiosis a progressive disease? I think that in this slide and in ensuing Roentgenograms, I can say and show you that it is. If you notice, this first slide, incidentally, all of the whole lung sections in pathology which I am showing you today have been prepared by Doctor Goffe in Cardiff, Wales and he lent me these tissues so I could demonstrate them to you.

If you notice here, in this first whole lung section, there are coal macules and in this lung we find no evidence of focal emphysema. However, in another case which had been a coal miner for a longer period of time, here you see the generating coal macules and focal emphysema and some coal macules maintain themselves without degeneration.

Now, what the contracture of the coal macule is due to is only a matter of conjecture. It may be that the impure fractions in coal dust besides carbon, have a fibrogenic tendency and cause this contracture over a long period

or else it may be due to the mechanical effect of the macule and the effect on the macule of the surrounding tissue. Perhaps this man had some silica in his coal macule, just enough to cause the fibrous contracture over a long period of time.

And now you see, in this final section here, all of the coal macules have disappeared and the entire lung field represents a Swiss cheese affair of focal emphysema. This, I believe indicates pathologically at least, that the disease is progressive, and you can see the progression in one slide, that the middle lung section - within that one section, you can see the focal emphysema and the macule before it has degenerated. While we're looking at these sections, we may see the reason why so many of the Roentgenograms appear to have only increased markings.

Of course, most of this, most of the macules and the tissue, as we see it, is not radiopaque and, therefore, in a large - in an X-ray of the entire lung, you have what may be called a little out effect, increased areas of translucency and increased areas of opacity, but they neutralize themselves when they finally come to the X-ray screen and that's why we see only evidences of linear markings, and of course, some of these linear markings may be ascribed to what Doctor Vorwald pointed out this morning in his microscopic sections.

Next slide. Now, this is a man who left the - left his coal mining occupation in 1948. If you notice the first X-ray, this was taken in 1949. Notice the type of X-ray he has. At this time, the man had already left his occupation in the coal mine and had no longer been exposed to any coal dust.

However, in the second X-ray taken one year later, you see that he has developed conglomeration and coalescence in areas where previously it was only suggested, that it had just started and then finally, in this last X-ray, another year later, you see that he has had additional progression of his disease.

Now, whether the progression of the disease in this case is due to the effect of the coal dust upon the tissue, or whether it is due to the fact that he had a silica admixture in the dust that he breathed, or whether it is due to superimposed infection, is not of material concern. The fact of the matter remains that this man removed from his occupation, showed a progression of disease in his lung fields which may be ascribed, directly or indirectly, to the basic pneumoconiosis.

Next slide. Now, in dealing with these patients, we find certain complications which are more frequent than others and if you notice the first complication listed is emphysema, the second one, bronchitis and bronchiolitis,

and then below I explain those two.

Emphysema and bronchitis are so constantly associated with clinically significant disease that they should be probably considered part of the clinical entity rather than complications. Now, we also have recurrent pneumonia in these patients, cor pulmonale is very prevalent with associated cardiac decompensation. As a matter of fact, if the cardiologist would examine people who suffer from pneumoconiosis, they would not regard cor pulmonale as an unusual entity, but would find it not only has acute manifestations, but we who are interested in dust disease of the lung find it is quite prevalent as a chronic disease just like hypertension.

Bronchiectasis, we have found in a substantial number of our patients; tuberculosis is, of course, one of the complications. Spontaneous pneumothorax, we have observed about twenty to twenty-four patients; I don't know the exact number, in our series, and I think that's a higher incidence than you should find in the control population group.

And, finally, pulmonary osteoarthropathy, we do find and I put that down as a complication because of the manner in which you can see dramatic results, when you have this hypertrophic pulmonary osteoarthropathy, if you treat the man's lungs, clear up the bronchiectasis, the bronchial

edema and infection, and don't give him any aspirin for - or any specific measures for his arthritis, so frequently is there a manifestation of improvement in his pulmonary function, general pulmonary function, the effects of the pulmonary osteoarthropathy is gone, with few exceptions for inclement weather and so on, as long as you maintain a rather satisfactory situation in the man's lungs.

Next slide. Now, this is an example of a patient who had pneumonia and then - now, of course, this man expectorates at a rate of sputum daily that I thought he had bronchiectasis, but you can see from the bronchogram, it is within normal limits and in the right lower lobe where it doesn't fill, probably the result of accumulated secretions, indicating that these people do have an entity which may be confused with bronchiectasis and has to be differentiated from the bronchiolitis which is associated, probably responsible for the bronchospasm and the bronchial edema, and a lot of these - a lot of disability, which one sees in these cases.

When you clear up the bronchiolitis, these patients have experienced a great deal of relief, and are able to do many things that they haven't been able to do previously.

Next slide. Now, this is an example of what we call temporarily disabling pneumoconiosis in soft coal

workers. In his lower left lung you'll see he has pneumonia and then there you see about a month later or so, that the area of pneumonia has cleared. Now, this patient, when we first saw him was critically ill, and such is the case with all of our patients who develop secondary complications. They live in such a delicate cardio-respiratory balance that a supervening, intervening infection, pneumonia or systemic infection throws the weight of balance against them, and these people become critically ill and disabled.

However, when they're treated and they get over their illness, they return to their previous status, perhaps with some additional damage to their pulmonary tissue which we can't measure and want to resume their work.

Incidentally, this patient is back at work in a coal mine and does not want anybody to know about the fact that he has pneumoconiosis because if they find out, they will take him off his job, which should be done. He has been advised to leave his occupation, but we as doctors, have no right to say to a man, 'You get out'. It's up to him to make the final decision, because he's the one who has to survive.

Incidentally, I only want to mention one thing about the treatment of these patients. You can not rely upon the parental administration of antibiotics and other agents. These people, to obtain the best results

therapeutically, I don't care what you use as long as you use something that's indicated and that has been successful in your hands. Parental therapy with penicillin, for example, in a case like this, would prolong the patient's convalescence maybe two or three months before you find that he is able to go to work, whereas with aerosol therapy, you attack the disease directly either by hand or with intermittent breathing pressure unit designed by Doctor Motley. It doesn't make any difference, but remember, the aerosol route, the direct approach to the disease.

Next slide. Now, this is an example of an individual with cor pulmonale. Notice the lazy character of the changes in his lung fields. He worked in a coal mine for thirty-five years. He is seriously disabled and he is unable to take a bath.

Incidentally, I'd like to point out that in our clinical experience, we find two very important things to ask patients, because sometimes a patient comes to you because he knows that the disease is compensable and he comes with the idea that you're going to establish the diagnosis and he comes in complaining of shortness of breath, cough, can't work, can't sleep, can't do nothing, is mad at his wife, nervous, can't do anything, but there are a few things you can find out.

For one thing, these people can't take a bath in

hot water. If they take a lot of water, and if they really have pulmonary disability on the basis of their pulmonary disfunction, they become more short of breath and have to be helped from the tub. That's due to peripheral vasal dilitation and increased oxygen demand.

Secondly, these people are always more short of breath in the morning than they are at any other time of the day except when they're working, so after they've had a good night's rest, when they wake up in the morning, if you ask them how do you feel, if they're really suffering from a dyspnea due to pulmonary disease, they'll tell you 'Doctor, I didn't sleep well and when I got up this morning I didn't feel well; it took me about an hour to stir around and start feeling better'. That's a good differential diagnostic point which we should keep in mind and it would also help us to differentiate between cardiac disease - dyspnea and pulmonary dyspnea.

Next slide. This is an example of cardiac failure in a patient with pneumoconiosis and before you - and before you label this patient as having pneumoconiosis, you should correct his heart failure first and then re-examine his roentgenogram to see if he still has evidences of pneumoconiosis. Here you can see the enlarged cardiac shadow on the left with increased markings of nodularity, and then here on the right you find that the cardiac shadow is

much smaller but he still has the changes in both lung fields compatible with pneumoconiosis.

In the management of these patients, it is important to remember that you do not treat these patients with cardiac failure the way you do someone who has cardiac failure on the basis of rheumatic heart disease or arterial sclerotic disease. In these people, it is important to correct the deranged pulmonary function and make some attempt to correct first before you digitalize him, otherwise you are apt to create a load on the right side of the heart and precipitate a fatal accident.

Next slide. I don't know how this got here, but it is. Would you mind taking this out and leave it for last. This is a joker. Now, this is an example of a patient with pneumoconiosis, heart failure and bronchiectasis, all of those things in one patient. Notice the dilated bronchi in the lower lobes and notice the cardiac failure in the middle picture. This patient is still living. He lives in an oxygen tent, has been living in an oxygen tent for over two and a half years.

Next slide. Now, this is an example of chiefly linear increased markings with some evidence of nodulation and a spontaneous pneumothorax on the left, on the left-hand side. Now, when I say a man has linear pulmonary dust disease, or when I say a man has nodular pulmonary dust disease

or mixed, I mean that it's predominantly linear, that it's predominantly nodular and when it isn't predominantly one or the other, I call it mixed, because as a clinician, we have to have a simple method of approach to this problem that you can present to other doctors in the community, to doctorw who work in coal mining camps, so that they can work with you and understand what you're trying to accomplish, and when you get too scientific with them, when you get into the category of too much research, with a little doctor who works in the country, you lose all of his cooperation, so we have to give them something that we can see and that's simple.

Next slide. This man lived a little over three years, a little over three years in an oxygen tent and then died looking like a skeleton and if you see, in this lung field, he has evidence not only of silicosis, but also over here you see evidence of anthraco -- excuse me - of pulmonary dust disease of coal miners, with focal emphysema and macule, -- habit will always come out.

Now, next slide. Now, here you see the X-rays of two individuals who have about the same extent of pulmonary change in the Roentgenogram. Now, the sixty-four dollar question, how can an X-ray man tell me which one is and which one is not disabled? But just to speed things up, I'll tell you that the man on the left is disabled and the man on the

right is not. However, I want to tell you that subsequently the man on the right has developed disability, but at the time of this examination, he was not disabled.

Next slide. Now, this is an example of extensive changes in the pulmonary field, in a patient forty-five years old, twenty-nine years a coal miner, no disability. Nevertheless, look at the extensive changes in his lung field, by comparison with some of the other Roentgenograms which you have seen both in this lecture and in the other lectures which preceded mine.

Next slide. Here is an example of a seventy-year old man with an elevated - with an elevated leaf of the diaphragm on this side, resulting from pleurisy, pneumonia, several years previous to this Roentgenogram. He is seventy years old; he works; he worked in a coal mine at the time this Roentgenogram was taken and he had absolutely no disability that we could find, either - either in the clinic, on clinical basis or in a laboratory.

Next slide. Now, this is an example of tissue reaction. These two boys worked alongside of each other for fourteen years. This man is thirty-six years old, I think, and this one is thirty-four years old. Notice the difference in the way these tissues have reacted. Here you have a nodular variety of change in this man's lung field and in the one on the right, has chiefly a linear change in his lung

field, indicating in this case that two people of different stock, working in the same occupation, reacted differently.

Next slide. Now, here is an example of two boys who worked alongside of each other, I think, for thirty some years. You notice the similarity of their Roentgenographic changes.

Next slide. Here are two other brothers, both with negative tubercular tests, both with negative tuberculin test, and we can't prove tuberculosis. They worked nineteen years together and they've worked about ten years apart and you can't, well, you know, you have to say they look alike anyway, and it's just remarkable to see the similarity of the changes in these two X-rays even though they worked ten years at another occupation.

Next slide. Now, these two, these two brothers are the first of four brothers. Now, notice how similar these two X-rays appear. These boys worked together forty-one years. Notice the similarity and the change in these Roentgenograms, but they're brothers, and now their other brothers worked in another part of the same coal mine for thirty-one years together. Notice the man on the right with the conglomeration and coalescence and the one who has only worked about fifteen years already beginning to manifest the changes of his brother alongside of whom he worked.

Next slide. You got the last one - you got the

one you put up the first time - I asked you to put it last? And now, just to confuse you, as well as I'm confused, this is two brothers. These two brothers worked alongside of each other -- excuse me, not alongside of each other, they worked for thirty-seven years on opposite sides of a railroad car. They did loading and the reason they worked on opposite sides of the railroad car were because they loved each other and they wanted to be sure in case a rock would start falling on the brother, they would see it and they'd run over and save the brother.

Look at these Roentgenograms. See how different they are. Worked on opposite sides of a coal car, so therefore, even though Doctor Lanza is much older than I am, I'm going to improve on his terminology. I'm going to say from now on when you talk about pneumoconiosis, you have to talk about pneumoconiosis in soft coal workers in Alabama in Jefferson County in Mine Number 9, Shaft Number 8 on the right-hand side of the railroad track, and that's about what it comes down to.

Now, in conclusion - in conclusion, I think I have about two minutes left, is that right, Doctor Sander?

BY DOCTOR SANDER:

One.

BY DOCTOR FRIEDMAN:

That always happens when you deal with Yankees.

In conclusion, I'd like to remark as follows: That there is a disease in coal workers, that it's due to their occupation, that the disease, once it manifests itself clinically, is progressive, that different people may react differently to the same exposure of dust over the same period of time. As a rule, I have sixteen pairs of brothers - that's why I say as a rule - as a rule, brothers will react similarly, but you can't depend upon it, because I just showed you.

(Applause).

BY DOCTOR SANDER:

Well, as you can see, Doctor Friedman has had a considerable clinical experience with this coal miners disease. I have asked Doctor Fletcher to open the discussion of Doctor Friedman's remarks, and then I would - I'm going to ask for questions from the floor after Doctor Fletcher has finished his discussion, and I hope you'll have a good many questions to shoot this way.

BY DOCTOR FLETCHER:

Doctor Sander has said that I could make one preliminary remark on my paper this morning, which I neglected to say this morning. I suggested that if we could abolish tuberculosis in mining areas we could abolish the worst effects of disease. That might be taken to mean that pneumoconiosis is a form of tuberculosis. I just want to say that any such interpretation of my remarks is the exact opposite

of what I mean. The expression of this disease is by dust suppression. It is an occupational disease; it is not tuberculosis, but if the utmost human endeavor fails to reduce dust levels, that in which no radiological activity prevails, then the lessening of tuberculosis on this disease, might be lessened by strict tuberculosis control in this area. I am glad I have an opportunity to remove any misapprehension in regard to my remarks.

In regard to Doctor Friedman's excellent paper, I really want to sympathize with him from the bottom of my heart, because I started studying this disease as a clinician and it wasn't until I had been at it as a clinician for sometime that I managed to get my epidemiological colleagues to show me something about the disease.

As a clinician one is grossly limited. He tells us that he sees spontaneous pneumothorax as a common complication of this disease. That means to say that patients who have spontaneous pneumothorax with this disease come to his clinic. It doesn't tell us anything about the frequency of that complication in the community of men with pneumoconiosis, and we clinicians are at a gross disadvantage in studying occupational disease in that we can not resist analyzing our figures and thinking that they may mean something. It's a temptation that I have had to strenuously resist myself, under the most tremendous barrage of criticism

from Doctor Cochran, my epidemiological colleague, who thinks of clinicians with - as one of the lesser breed of rodents.

So I just want to sympathize with Doctor Friedman and congratulate him that he has derived so many interesting conclusions to the severity of this disease. I would just like to quarrel with him about this temporarily disabling pneumoconiosis. He says he has a man with a pneumoconiosis with pneumonia and he was grossly disabled. Well, I have seen that in London. He was disabled when he came in. I think he had disabling pneumonia, whereas you might call it temporarily disabling pneumoconiosis.

I want to quarrel with his suggestion that the coal macule is not as opaque as the silicotic nodule. The coal macule is smaller than the silicotic nodule. I believe the radiopacity is at great as collagen and the silicotic nodule is larger than the coal nodule, displaces more air and is, therefore, more visible.

And lastly, I want to ask him one very important question, that is in this tubercular negativity of cases of massive fibrosis. That we have never observed. We have found cases of massive fibrosis that only react to one in a thousand tuberculin and I believe we have had one go up to one in a hundred but never a complete negative, and I would like to know what strength of tuberculin he used for

that negative test. Thank you very much, Doctor Sander.

BY DOCTOR SANDER:

Thank you, Doctor Fletcher. Do you want to make your remarks now? Doctor Vorwald has a few remarks to make to correct a false impression of this morning, right?

BY DOCTOR VORWALD:

Yes, I should like to do exactly what Doctor Fletcher did, so that we do not leave a false impression with you.

First, in showing the slides stained for elastic tissue, Doctor Hussey called my attention to the point that I did call it elastic tissue rather than reticulin within the lung, and so I wish to correct that.

The second correction which I should like to make, being a correction, at least attempt to modify the impression which I left with you, and that is with respect to this focal emphysema and I demonstrated a series of cases of workers exposed to dust other than coal dust, where there was focal emphysema, and called attention to the fact that this focal emphysema was then not unique for the coal miners' lung.

In presenting these cases, such as from the granite worker, and from the limestone worker and from the graphite worker, I wish to impress upon you that those were the very isolated cases, that it has certainly not involved

the large group of individuals supposed to be exposed to these dusts.

Furthermore, with respect to the magnetite worker and the hematite worker, the incidence of this focal emphysema, about the foci of dust, is present probably to a greater degree than is present in the graphite worker in the other cases, and to a lesser degree in the coal miners' lung, but that does not mean that with the deposition of graphite in the lung, with the deposition of magnetite, with the deposition of hematite, and indeed with the deposition of limestone, that there must also, therefore, be present a focal emphysema and thus, therefore, the man - the patient must also have a respiratory difficulty. I think that is fallacious and I don't wish to leave you with that impression.

If I may go on and comment just a bit about Doctor Friedman's remarks, certainly Doctor Friedman has had a great deal of experience with the coal miners. I should like again to take a bit of exception with him on the - his statement with regard to the matter of progression, that it is of relatively little concern. He mentioned that it is of little concern whether it be due to massive local concentration of dust or whether it be due to a complicating infection.

I think it is of concern. We must know whether

these coal miners' lungs are more susceptible to infection than is the lung of a worker with deposition of other kinds of dusts.

Again, I should like to make a slight comparison as did Doctor Fletcher, that is, he spoke of emphysema, and that it is important to know the incidence by age also of emphysema, in the entire working population of the coal miners, and to compare that incidence with the incidence in the non-exposed population, by age and by racial characteristics. I think that is also very important before we conclude that emphysema, in the coal miners, about which he has seen, as a common denominator of all of the coal miners exposed and that it is higher than the incidence in the non-exposed population.

With respect to his last remark, that is that he will not only have to know whether these coal miners come from Alabama, and whether they come from Jefferson County and Mine Number 9 and the right side of the track, I think that's precisely what he has to do, and we all have to do that, and furthermore, I should like to know, and I think we have to extend it, we want to know whether he comes up the track or down the track. We want to know whether that track is outdoors or indoors.

I think those are the things that we must know if we are to solve some of the problems relating not only to

the pulmonary changes in coal miners, but also the pulmonary changes in workers exposed to other dusts.

One more comment and that is with respect to the Roentgenogram and the pathological manifestations of the disease; it is true that the pathology, the pathologies, - the pathologist frequently sees more disease in the lungs than manifested in the chest Roentgenogram. But I don't believe that that should be used as an argument to support the view that there is always something more in the lung than is present in it, than is visualized in the Roentgenogram. Should we follow that reasoning, then we could all - we should say then also, well, this Roentgenogram is negative, it shows no evident change, but there is something more in the lung than what we see in the Roentgenogram. Therefore, this man must have pneumoconiosis, because he had an exposure to coal dust, so we throw the Roentgenogram out the window, we throw the physical examination out of the window, and all we want to know is did or did not this man have an exposure to dust. If he did, then he belongs to the category of pneumoconiosis; if he had no exposure, then we may question. Thank you.

BY DOCTOR SANDER:

We already have a few questions of Doctor Friedman. I'm going to ask one and then I hope we'll have some more from the floor and then we'll give him a chance to

answer them.

I'd like to know how he makes a diagnosis in a man who has been a miner for some twenty-five years, whose chest film at age sixty-five, when he's retiring, shows only emphysema, nothing beyond that. What - how does he go about making the diagnosis beyond the hot tub test and the - the history of his not feeling well when he gets up in the morning? What do you have besides his history, and possibly the lung function test, which undoubtedly will show a low maximal breathing capacity, because he has emphysema? Anyone who has emphysema has a low maximal breathing capacity. At age sixty-five it's lower than it was at age forty-five, and if you have the additional emphysema, it's going to be still lower. How does he make the diagnosis of coal miners' pneumoconiosis with that situation in mind?

Do we have some other questions? Doctor Kline?

BY DOCTOR KLINE:

Doctor Friedman, I didn't come in at the very beginning of your paper and if you've answered my question, I'm wasting your time, but you talked about cardiac failure in these cases, and you mentioned that before one gave digitalis or treated the patient with the other methods that we have available for the cardiac failure, one should do something about the pulmonary condition. I presume you were referring to McMichael's work in England on the effect of

digitalis on the veins of the body. This work has not been widely accepted, and not generally confirmed, but I'm not taking any exception to that. I am asking this question. What does one do about the primary pulmonary condition? The disease of the heart is the result of increase in the pulmonary artery and the right ventricle. Do you have some therapeutic tool which corrects this condition before correcting the cardiac disease?

BY DOCTOR SANDER:

Any other questions?

BY DOCTOR AMBERSON:

What was the time element between the last X-ray film and the last post mortem examination? -- Doctor Amberson -- This calculation effects me as batting about ninety-seven percent, which is pretty good in any league. The time element between the last chest film and the post mortem examination.

BY DOCTOR SANDER:

In which case?

BY DOCTOR AMBERSON:

I think there were two or three.

BY DOCTOR SANDER:

He had two or three. Any other questions?

BY DOCTOR BRISTOL:

I'd like to ask Doctor Friedman if he thinks it's

possible from the X-ray to make a diagnosis of focal emphysema, number one, and X-ray alone, that is; and number two, do you believe that emphysema is a complication of simple coal workers pneumoconiosis?

BY DOCTOR SANDER:

Any other questions of Doctor Friedman?

(No response).

You have the floor, Doctor Friedman.

BY DOCTOR FRIEDMAN:

On my way up again. I should have stayed home. I'm really surprised. I thought it would be worse than that. I don't know whether I'm going to answer these questions in order and I don't even know whether I'm going to answer them, but I'm going to say something about each one.

As regards Doctor Fletcher's remarks about the incidence of spontaneous pneumothorax, that's true, it's clinicians' incidence of the disease and in my particular situation, I am not in a position to compare a comparable control group of coal miners whom I study, because the people who come to see us, sick coal miners, we don't get a cross-section of the coal mining population.

However, I do think, Doctor Fletcher, that the incidence of spontaneous pneumothorax, even without the comparison, appears to be higher than in the normal population group. Of course, I know that Doctor Goffe, and you

come from that school of Doctor Goffe, feels that spontaneous pneumothorax probably is unrelated to the disease because of the location of the blades, and when I made that statement, I knew I was on dangerous ground. I thought you'd overlook it.

Regarding the tuberculin testing, we use one to one thousandth to begin with and we end up with one to one hundredth and if you're ever down in Alabama, I'd like for you to come down and see some of our negative skin tests, because they really complicate the picture when we can't obtain a positive reaction, and we frequently test them again in three months or six months.

And then, about the location, then Doctor Vorwald made a remark about the incidence of emphysema in coal workers. I took one hundred patients from the age forty to sixty in our own clinic, random sample, men, and then I took a group of coal miners, forty to sixty years of age, and compared the incidence of emphysema in both groups.

Now, I admit before I go any further, that it's not a fair comparison because the people in the control group who come between the age group of forty and sixty, came in complaining perhaps of some heart disease or gastric disturbance or something of that nature, but the coal miners all come complaining of something wrong with their lungs, but at least in that group, I would like to give you the

benefit of what we found.

We found less than ten or twelve percent emphysema in a control group whereas in a group of coal miners who came to be studied, and we used maximal breathing capacity, both lung capacity, fluoroscopic examination, X-ray, we didn't use any gas studies, we're not equipped for it - we found that the incidence of emphysema by the most rigid standards in a coal mining standard, in the age group of forty to sixty was in excess of seventy-five percent.

I admit, to begin with, that that's an unfair comparison, but as I say, I don't have a cross-section and I wasn't able to X-ray all of the coal miners in an area whether they were sick or whether they weren't.

I'm glad that Doctor Vorwald felt that the location of the man's work was important. We all realize that, and I think we have to realize, not only the geographical location of the mine, but where he works in the mine, whether he's a timberman, whether he's a short fire man, whether he does drilling or whether he does overhead pinning, these are all important factors, and those of you who may not be familiar with some of the more modern methods of coal mining, I would like to tell you, and Doctor Fletcher made some remark touching on that this morning, in this machine age of mining we're beginning to see patients in the lower age groups who, after lower periods of exposure as coal workers,

are developing pneumoconiosis by Roentgenographic change and functional changes. It's because the machines don't know to cut through just coal, they cut through everything and they want to do that so you can get the most out of a seam of coal, and the overhead pinning, that is, of course, the same thing as drilling in pure rock, and you're going to get a pure silica exposure because the purpose of overhead pinning is to fasten the roof of the coal mine and instead of with beams, you're going to fasten it to the overhead strata.

I had the privilege last week to examine some coal miners in Peru, South America. I'm only sorry that they wouldn't let me have the X-rays to bring up here, because what we see in our coal mines isn't anything like what we see there, what we see in their coal mines isn't anything at all like what we see in our coal mines in Alabama and Doctor Fletcher didn't show anything that looked like any Peruvian coal miner, and if you think we have patients, they have many more, because their patients are Indians who live fifteen or sixteen thousand feet above sea level, and to begin with, they have emphysema and then you have to start to evaluate the disability on the basis of that, so we ought to be pretty glad our problem isn't complicated in that manner.

Now, Doctor Sander asked me, I think I have the

age right, in a man about sixty years old, who has worked so many years in a coal mine, who has no Roentenographic changes, but just has evidence of emphysema by our standards, how could I say this patient had pneumoconiosis. I wished I could say so, Doctor Sander, because I'm convinced in my own mind that many of these people do not show Roentgenographic changes compatible with the results of their occupation. I believe that they develop emphysema or rather we should put it this way, I believe just like another sixty year old man, they may develop emphysema in the usual course of life, but they're more prone to develop emphysema if they've worked in a coal mine.

The mechanical effect over a long period of time of the inhalation of non-soluble dusts, the effect in internal bronchial, with infection and without infection, causes an intermittent occlusion over a long period of time of a balance value of nature, and if you do that long enough and often enough for twenty to thirty years, you're going to have some effect on your alveolae, and you're going to have emphysema as a result of your occupation.

The day will come, I'm certain, that we will be intelligent enough as doctors, and will find the correct diagnostic methods to be able to prove that the emphysema we see in coal workers, even without Roentgenographic changes, may be the result of their occupation, taking into

consideration the fact of the usual aging process in man.

Now, Doctor Kline asked me about treating the patient with congestive heart failure and something about why we didn't use digitalis first, and what corrected the pulmonary function before we treated heart failure. I'm not familiar with the man's name you mentioned, that's only my own observation.

We found that we are able, very quickly, to improve the pulmonary function in an individual with the judicious - or judicious use of proper aerosol therapy and oxygen. We treat them for about twenty - about twelve hours for their lungs first. We give them aerosol therapy, oxygen, and then after we have improved their pulmonary function to some degree, we then go ahead and digitalize rapidly with one of the popular new preparations that are designed for rapid digitalization.

The reason we did that was because we had occasion to treat several patients with pneumoconiosis and heart failure and we treated them with the standard methods and we lost a few patients, so we felt that we would give it a try and treat them from the other end first, treat the pulmonary side first. We did and we found that it's been successful.

Doctor - Doctor Bristol asked me how do we establish the diagnosis - will you restate your question; you

know I had a lot of trouble here keeping up with you fellows.

BY DOCTOR BRISTOL:

Well, I believe you mentioned in your paper, that in demonstrating one of your chest Roentgenograms, you pointed to an area of focal emphysema and I asked you the question, do you think we can see focal emphysema on the Roentgenogram.

BY DOCTOR FRIEDMAN:

Well, now, I get it now. Now, when you see a lot of X-rays in coal miners, it's just like anything else in medicine, you get used to seeing something, you think you know something and after a while, when you've had autopsy specimens to go with Roentgenograms to compare, and then you can go back and compare your Roentgenogram with your pathology, then you can see some changes in another X-ray which at least suggest the same thing you saw previously, the diagnosis of focal emphysema is not a definite diagnosis from the Roentgenogram; it's presumptive on the basis of experience.

BY DOCTOR BRISTOL:

Well, would you enlighten me by telling me what characterizes, in your own mind, focal emphysema in the chest X-ray?

BY DOCTOR FRIEDMAN:

Well, in the chest X-ray of focal emphysema, you

start to get the idea you're looking through something like a mesh work, like a dilated sponge. You get the idea that the areas of translucency, surrounded by areas of increased density have a regular nature, but you can not decide, it's a sort of a linear character of the X-ray, you can not make the diagnosis from the Roentgenogram, and it's a presumptive diagnosis, and after you've seen enough of them, I think you can usually guess correctly.

Now, another Doctor asked me about the - about the time element between the Roentgenograms and the necropsy material, is that correct? Now, the Roentgenograms perhaps were thirty - a month before the patient died or a week, it doesn't make much difference, but just within that limit, I think, with the exception of one patient who died, the one with the spontaneous pneumothorax. He didn't die until about two and a half years after we had that X-ray, but I wanted to show the spontaneous condition.

Now, the radiologist didn't have a great deal of difficulty batting ninety-eight or ninety-nine percent, because most of the X-rays that he looked at were pretty easy to diagnose as pulmonary dust disease of coal workers, together with their occupational history. However, as Doctor Bristol pointed out in his question, the X-ray man was not able to tell me, nor am I able to tell you definitely that this man, this patient was going to have focal emphysema.

in the pathologic specimen. It's presumptive on the basis of experience.

BY DOCTOR BRISTOL:

Louis, did you forget my question which was, is emphysema a complication of simple coal miners pneumoconiosis?

BY DOCTOR FRIEDMAN:

Emphysema is a complication and, as I said ...

BY DOCTOR BRISTOL:

Simple now.

BY DOCTOR FRIEDMAN:

Emphysema is a complication of simple pneumoconiosis, focal emphysema, but you see bronchitis, emphysema and chronic bronchitis are so closely associated with pneumoconiosis that you should consider the part of the disease entity. You remember I showed the pathologic specimen of the degeneration in the individual, and you saw that that was a definite change in the pathology and Doctor Vorwald demonstrated that this morning too.

BY DOCTOR SANDER:

We're - thank you very much, Doctor Friedman. We're a little bit late, but I think a two minute stretch would help all of us.

(Recess taken from 3:30 to 3:50 P.M.)

BY DOCTOR SANDER:

Will you take your seats please? We have two very interesting papers remaining this afternoon, the first one by Doctor Hurley Motley, recently of Philadelphia, who has done the most, one of the most outstanding pieces of work in lung function studies and therapy of pulmonary dust diseases. Doctor Motley is now heading for a more salubrious title. He is on his way to Southern California as Associate Professor at the University of Southern California Medical School. Doctor Motley.

BY DOCTOR MOTLEY:

(Doctor Motley read a prepared paper which is on file at the Saranac Laboratory).

BY DOCTOR SANDER:

I think you'll agree that's a lot to digest in forty-five minutes. I don't see how you could cover that much that fast. We'll withhold discussion on Doctor Motley's paper until after the next paper, because they're on essentially the same subjects.

Doctor Philip Hugh-Jones comes to us from England. He has been with Doctor Fletcher's pneumoconiosis research in Cardiff. Doctor Hugh-Jones wants me to state, asked me to state that his collaborator, Doctor Gilson, has worked with him and would be one of the speakers had he been able to come here today. Doctor Jones will give us his experience in the function studies of the coal miners in South Wales.

Doctor Jones.

BY DOCTOR HUGH-JONES:

(Referring to an exhibit on table) Just for the benefit of any security people, that isn't part of an atomic bomb.

Mr. Chairman, Ladies and Gentlemen: The lung function studies I want to talk about this afternoon, as the chairman has told you, were done in collaboration with Doctor Gilson, and we were working under Doctor Fletcher in South Wales where this extreme amount of coal workers pneumoconiosis has occurred.

Now, the most important symptom of pneumoconiosis is excessive breathlessness on exertion. But since this symptom is not specific in any way and neither are there any specific clinical findings of the disease, the diagnosis of pneumoconiosis in life, depends on the radiograph, and all too often, the disease is thought of as a radiological abnormality. However, I know you would agree with me that it is the disturbance of lung function and not the radiographic changes which matter to the man, and the main purpose of our work was to find out the functional significance, if any, of the radiograph.

But before I describe that experiment, I would like to be quite clear as to what this 'coal-workers' pneumoconiosis' is that we see in Britain. Radiologically

and pathologically, it's distinct from silicosis, that is the pure classical silicosis, and we know, for example, from the work of Gough, who found it in the coal-trimmers at the docks, that it does occur, just from the exposure to coal dust only, because those men who load the coal in ships have no exposure to rock dust, and they get typical X-ray findings of coal workers pneumoconiosis, and in the mines, it usually occurs in the worker on the coal face.

Now, the disease we see in Wales, radiologically, is no different, as far as we can make out, from the descriptions of the disease given here in the United States, given in other parts of Great Britain, given in Europe and given in Australia, so I want to be quite clear that I'm talking about coal workers pneumoconiosis, and the results I think are applicable to it in Wales certainly, probably in other parts of the world, but we have no reason to suppose that the studies are applicable to silicosis, which is a different pathological condition.

Now, in describing our work, I shall use the International Classification of the X-ray which Doctor Fletcher told you about this morning, and may I have the first slide please.

And, as you remember, from his remarks, that is divided into the simple pneumoconiosis. There is a typical example of the stage tree of the simple disease, and into

the complicated or conglomerate shadows. May I just have the next two slides. The first one there is a typical Stage B, and lastly Stage D, just to remind us what we're talking about, which shows the extreme distortion -- may I have the next slide, please -- the extreme distortion you get of the lung structure.

Here is the slide, with the trachea moved right across and this large basal emphysema which I think, radiologically, everybody would agree is basal emphysema.

Now, for our work. The main intent, as I have said, was to find the functional significance of the different X-ray stages, but we also wanted to know why coal workers were breathless. May I have the next slide. So I can put down that was our first object of our experiment, the cause of the breathlessness and its severity in relation to the radiograph.

Now, the second intention was a more ambitious one, and that was to try and find out the interrelation of the different lung function tests we were using, that is to what extent one test overlapped with another, and what precisely they were measuring in the lungs.

Now, I shall deal this afternoon, with our first object and the second I will put out as a demonstration for the results of that. Now, of course, many other workers have tried to get out this first object, but a review of

the literature has shown us that very few people claimed that there is much relationship between the radiograph and the functional state of the man. But also a review of the literature convinced us that workers who have claimed to have found this lack of relation had very little real power to make that conclusion.

And I have put up here, and I hope you'll forgive me, what I think are the necessary criteria before one can accept any work relating the X-ray to the functional state of a man. First of all, it must be an unbiased sample and a hospital population is particularly unsatisfactory from this point of view, because obviously, the breathless man comes up to the hospital, and I would disagree with Doctor Motley when he says that his patients were unselected. He may have not selected them, but when he draws a conclusion that there is no relation between the X-ray and the functional state of the man, I would submit that his work is done on what is, inevitably, a biased sample, if it were volunteers coming up to a hospital.

Secondly, there must be allowance made for other independent variables which everybody will agree effects that function besides pneumoconiosis. The obvious one is age, but there are many others.

Thirdly, there must be adequate functional tests and we must know their accuracy, their reputability, and

what I have called their discrimination, that is their power of segregating the normal from the abnormal. I will explain that later if you people are interested.

And finally one requires precise radiological description. My own impression of the literature is that physiologists have gone to a great deal more trouble over their physiological tests than they have over the radiographs and there are very few physiologists who are meticulous enough to accept carefully graded tests to give the same criteria of excellence to their radiographic selection.

Well, bearing those points in mind, we designed the following experiment -- may I have the next slide, please -- which was designed to try and meet those requirements. We selected, at ages thirty-five, forty-five and fifty-five years approximately, eight men in each of the different radiological categories, normal working man, that is men with less than simple pneumoconiosis, men showing Category 1 or 2, men showing Category 3 or 4, B and the D's.

The men were selected entirely on their radiograph, on no other basis at all and the radiographs were classified and read in duplicate, by independent observers. Furthermore, we also selected, as another control besides the control miners, we selected forty men who were non-miners and we chose them simply to be sure whether mining

itself might have an effect on lung function as opposed to the radiograph.

Well, now, besides all these men, we did, as you will see in the talk, select some other workers as other controls or some other men. We selected five cases of advanced non-industrial emphysema, chosen for use by Professor Christie in London, who sent us them as undoubtedly cases of emphysema, and we also selected two rather rare cases of a curious fibrosis in the lung, where it was thought that there would certainly be a gas transfer defect. That is those other cases were simply put in as controls to make sure that the appropriate lung function tests did measure what they were meant to. That is, if we got negative findings in pneumoconiosis, we wanted to be sure that the tests themselves were not at fault.

Now, before the men were tested by ourselves, they were examined cardiologically and clinically, and the clinicians were two independent observers, who not only examined the men, all these men, a hundred and sixty-five cases, but they graded them according to their degree of breathlessness.

May I have the next slide, please? And their breathlessness was graded purely symptomatically on a series of questions as to whether they were undoubtedly as good as other men when they were walking about on the flat, whether

they were able to walk with men of their own age on the level, but they got behind on hills or stairs, whether they were able to keep up with men on the level, but they dropped behind after a long time, or whether they could only walk fifty yards or finally whether they were breathless at rest.

Now, such a classification is obviously open to observation rather, but when two independent clinicians did it and we have found that such a grading of breathlessness does at least enable one to define men into, shall we say, four categories of breathlessness with which to compare lung function studies.

Now, for our own tests. May I have the next slide please? I haven't got time to go through in detail all of these tests, but I think and hope they're fairly familiar techniques. The exercise test was a stepping test in which every man did work at the rate of 350 killogrameters a minute, and we measured the amount of air he needed to do that standard amount of work. We also, in some cases, measured the arterial oxygen saturation of the blood before and after the exercise.

Now, the exercise was, in fact, similar to the sort of exercise that one gets on a bicycle or a pedometer, in that it was independent of the weight or size of the man. That was standardized out. We measured, as you see there, maximum voluntary ventilation and we also tried to determine

whether a man had any bellows spasmor not, by using adrenalin.

We measured the arterial capacity, using our closed circuit, which compares very well with the open circuit technique described by Doctor Motley here and first put out by Doctor Kellner, and finally the last test in which we tried to find the intra-pulmonary gas distribution and the rapidness of gas transfer into the blood. I must spend a little time on that.

Now, our men, as I told you, were not volunteers coming up to the hospital. They were men selected at random from the whole of the coal field, many of them working miners, and we would, therefore, think, that it would unjustifiable to do arterial puncture on them, and so - and we wanted to try and get a test which would give us the information with as little as possible in the way of disturbance to the man, and we chose to give them an inhalation of air in which some of the nitrogen was replaced by helium simply to act as a trace gas, and in which there was a small quantity of carbon monoxide introduced.

The idea was that helium goes into the lungs and is distributed without going through into the blood stream. It's the less soluble of all gasses in the blood. The carbon monoxide on the other hand, goes across into the blood stream, and in lung concentrations, exerts negligible back-tensions. May I have the next slide, please?

You will see here the results, the net results of the test. Here I am plotting up here the concentration of gas that the men breathe out after he stops inhaling the mixture here. And you will see that by about forty breaths or fifty breaths, a normal subject is breathing out what he is breathing in in the way of helium. On the other hand, the carbon monoxide comes up and then flattens out. The difference here being due to the rate, due to the absorption of the carbon monoxide into the blood stream.

Now, the results. As you see, there, they are not very helpful and they do need mathematical analysis in the form and components of these curves, and I can't go into that now, but I would like to tell you that in normal subjects, the helium distribution curve in the blood and in the lungs is the sort of thing you would expect if it followed through the lungs. That is an exponential curve, and in emphysema, it seems, it ventilated much more quickly and washed out of gas much more quickly than other parts, so that instead of being a single exponential, it takes the combination of two exponential curves, and the mathematical analysis is entirely different.

It takes much longer for the man to replace the gas in his lungs and the replacement is in a different form, and we simply express that in terms of two mixing indices which I shall describe and they do represent the

degree to which the lungs are unevenly ventilated. That, I shall use later on, as a physiological definition of emphysema.

The carbon monoxide is expressed as the proportion of gas removed from the inspired gas, because that gets over the difficulty of the different ventilation of different subjects when expressed like that.

Now, for the results. May I have the next slide, please? Here you see the standardized ventilation. That is the amount of air the subjects need for doing a standard amount of exercise, and here are our different radiological groups and you will see that a-apart from a few men lying outside these lines, most of the men did not need a particularly increased amount of air compared to the normal subjects, to do the exercise.

In fact, in other words, they had only about a twenty percent rise, this is about twenty percent here, greater than the normal, in the most abnormal subjects, in their ventilatory requirements for a given amount of work. So it's perfectly clear that the cause of the breathlessness is not primarily an increased need for air when doing a standard amount of work, in pneumoconiosis.

If, on the other hand, you look here at the maximum voluntary ventilation or if you like, the maximum breathing capacity, that represents the maximum output of

the pulmonary bellows. It tells you how much air a man can pass in and out if he tries his hardest and you will see that, first of all, there is a very marked falling off of the maximum voluntary ventilation with disease, but secondly, there is a very wide scatter about this average and if you look at this diagram, these crosses represent the thirty five and the circles elderly men and you will see there is a big age effect. Mostly the young men are at the top here and the old men are at the bottom, so now if we take out this effect of age -- the next slide -- and you see the results now of the mean maximum voluntary ventilation of those different radiological groups against the age in years, you will see that normal men drop their maximum power of their lungs as they get older, certainly men with simple pneumoconiosis, but you will notice that simple pneumoconiosis or the degree of it doesn't make much effect.

The 3's are just as bad as the 2's and even the none, without pneumoconiosis, or shall we say without any radiological pneumoconiosis, are worse than the other men, but not seriously worse, but when you come to the complicated disease, you will see that they have a marked falling off of the maximum ventilatory power of their lungs, particularly in Group D.

May I have the next slide, please? Here is the result really shown in another way. It's the form of a

solid histogram. You will see the base of it is our original experimental plan. Here are our different age groups and here are our original - here are our different radiological groups on the bottom and the height of the groups represents the average blocks at the top. On the top of the clinician's effect of their breathlessness done entirely independently of the history, and the bottom it says 'dyspneac index used by McCann', in which we express the amount of air a man had to use as the amount of exercise in proportion to the amount he conceivably could use.

Now, it's obvious when you stop to think about it, if you're using all the air your lungs can take in and out, you're feeling extremely breathless whereas if you're only using about twenty percent of it, you're not feeling very breathless, and this index we do find agrees remarkably well with the assessment of breathlessness given by the clinicians, but it's a more sensitive test. It's not surprising, you can measure the thing better than you can - well, I wouldn't like to say guess, I'll say estimate it, and you see here that the normal subjects get slightly more breathless with age, but the important thing is that simple pneumoconiosis, although it puts a slight premium on normal aging, doesn't have much effect until the men are very old, that is, fifty-five years, but the complicated pneumoconiosis puts this marked premium on aging and

furthermore, this Group D, the rate of change with age is very much steeper.

May I have the next slide, please? Now, Doctor Motley expresses most of his results in the terms of residual air percentage of the total lung volume, and here I know you will forgive me, if I disagree with him to some extent, First of all, in some of his papers, and he did this afternoon express his other results in terms of this one test, and it does seem to me that automatically then, that test is labeled as a hundred percent and everything else is worse than that, and I don't quite see why he chooses that test for emphysema and not some others.

Secondly, I would like to show a difference in our results than the ones he got. Here is the residual capacity percent and you will see that in the pneumoconiosis cases, there is a rise, but not a very marked rise. There is a rise, you will note, with age in normal subjects and there is a slight increase with disease particularly, in the group D, but it's nothing like as marked as in the non-industrial emphysema people and the - there is one slight point here, and that is with regard to hospital populations.

When we took a hospital population, we found exactly the same results as he did, that at least fifty percent of the men had a large residual air percentage in that

case, whereas here only about nineteen percent had one in the general population with pneumoconiosis.

May I have the next slide, please? Furthermore, I would like to analyze the meaning of this residual air percentage. Here you will see the absolute figures expressed, and what's noticeable here is not so much the rise of the residual air itself, but the fall in the total lung volume, and I believe you're seeing in pneumoconiosis exactly the same thing by looking at the residual air percentage as you are in measuring the M. V. B. or the fall in the vital capacity.

The percentage in pneumoconiosis is, by and large, raised because of the fall in the complimentary air, whereas in some of the non-industrial emphysema, both the residual capacity is increased, and the total lung volume increases, so that the percentage is still up, but you may get a rise of residual air percentage from two different defects, one from an increase in the absolute volume and you get that, notice, in Category D pneumoconiosis, where I believe they are developing true hypertrophic emphysema on the radiograph, whereas in our people it looks to me as if it was due to a fall in their bellows, namely, their vital capacity, rather than a rise in the residual air.

May I have the next slide, please? Now, we've mentioned this term emphysema. Well, we might discuss

afterwards what we mean by emphysema, but the one thing is clear cut and it's been demonstrated very ably in Philadelphia in the States by Fowler and Comroe and his group there, that in this emphysema, you do get this inequality of mixing of gasses in the lungs. We find exactly the same thing, and this is the result of this helium mixing index which expresses the primary exponential - the binary exponential replacement of gas, and you will see that the normal binders, if I may call them that, are grouped with the normal subjects, truly normal. The simple pneumoconiosis are grouped with Category B, and it's only Category D that you get a very marked falling off of the index.

If you compare that with the advanced non-industrial emphysema sent to us from London, you will see that the test does show a marked effect in non-industrial hypertrophic emphysema. Now, it is tempting to suggest that this might represent what Gough describes pathologically as focal emphysema, because after all, there is no reason to see why a massive shadow per se, in this group B should alter the gas distribution in the lungs, and it is equally tempting to suggest that this corresponds with the large fuller emphysema of the radiograph. Anyway, we have not got enough pathological correlation to make that point clearly. Nevertheless, I think the results are suggestive.

May I have the next slide, please? Now, lastly,

what about the gas transfer defect in pneumoconiosis? Well, do you remember when I was talking about the exercise results, I did say that there was a rise of about twenty percent only, in the amount of air for a given amount of work and that would, to me, suggest an indication either of gas distribution in the lungs or gas transfer being deficient. Here are the results of the carbon monoxide uptake, and you will see that there is not a marked deficiency of carbon monoxide uptake until you get down to the Group D, and even then it isn't very great and when you remember the scatter about these averages, you'll find that the results here are barely, of bare significance.

These are the Category D as we have had before; those are the Category B and those the simple pneumoconiosis, but to show you that that test does reflect gas transfer defects when it occurs, here are these cases of rather rare fibrosis which have been published recently in the Medical Journals, and you will find that these men were - with curious fibrosis in the lungs, do take up about twenty percent of the carbon monoxide in the mix-up, whereas these people take up about seventy percent.

So I think you can say that gas transfer is not a marked effect in pneumoconiosis except in its later stages and then it is often associated with heart disease and cardiac failure in our experience, and I think that

result does fit in with the clinical observation that cyanosis is not a common feature of pneumoconiosis. It's breathlessness on exertion these men get, and not breathlessness at rest nor cyanosis.

Now, May I just try and draw some conclusions about this work, and to suggest to you some of its implications. First of all, it does seem to me that we have established on the average, that there is a relationship between the X-ray and the functional state of the man. Notice, I said on the average and in groups, and I think the reason why people have missed that relationship is because they have been working with bias populations and they have not taken the effects of age into account.

I don't know whether you remember that the slide I put on of the dyspneac index, did show that young men with - even with Category D pneumoconiosis, may not be very breathless whereas the old men may be extremely breathless, and if you read an X-ray in conjunction with age, you get an idea on the average, of the man's breathlessness, but that doesn't work in individuals, because the scatter about the average, and you do need functional tests to precisely say how disabled a man is, and no normal amount of reading of the radiographs helps you. On the other hand, the relationship to the radiograph, I think, is important from the point of view of prevention of the disease. We have shown that

it is the common disease which is the disabling disease, whereas the simple pneumoconiosis, whatever it's degree, and notice I say whatever it's degree, does not produce serious disability.

That gives one a guide as to how one can check the efficacy of dust suppression in the mines. It means that one can afford to let a man develop the very earliest stages of simple pneumoconiosis without thinking that he will be seriously disabled by it, and you can then say to the engineers, look, this mine is not safe and if the man goes on, on repeated radiological examination progressing, he will be liable to serious disability, by catching, if you like, or by developing progressive massive fibrosis and complicated disease, and the epidemiological studies have shown the complicated disease, whether it's due to tuberculosis or not, I don't know, but in itself, does not occur, and this is a significant and marked - at least Category 2 simple pneumoconiosis in the radiograph, so I think the practical importance of this work is in relation to prevention of the disease by periodic X-ray examinations.

Then, lastly, there is the question of compensation and disability, which I know many people will be asking. Well, first of all, there is the very intriguing and slightly worrying result that we found, that the normal miners, with less than Category 1 pneumoconiosis in their

X-ray, were significantly disabled compared with the truly normal non-miners, and that was confirmed in a pit where men were working underground and compared with non-working population, but obviously, that's got to be done on much larger groups of men in other industry, heavy steel industry, farming workers, shall we say, before we can make an assertion, but it is interesting that, shall we say, make a guess if you like, that breathlessness can occur in miners, compared with non-miners, to some extent, and one might argue that the disability is the thing on which the functional tests should give a precise answer.

The loss of faculty, if you like, is what the clinician has to assess, as the meaning of the functional tests and I think, personally, compensation should depend on that.

Lastly, I have said nothing about the interrelation of the tests which was the other object of our work. Now, Doctor Motley, for example, suggested that a battery of three tests, vital capacity, maximum breathing capacity and residual breathing capacity percentage, could form a useful measure of ventilatory function. It - perhaps I understood - or misunderstood Doctor Motley, but it did seem to me that he compared them individually with the sum total of the three. I would like to get his comment about that.

But what we have tried to do, instead of arbitrary selecting tests, is to try and find the precise mathematical relation to our problem and this rather curious looking object here which I have put out as a demonstration does show that. The angles between each of these knitting needles represents the degree to which tests are measuring the same thing. It's all right - this will be out on demonstration, you can see it somewhere else.

If tests are at right angles, it means they're entirely independent, measuring something to the - something totally unrelated. The nearer they come together, the more they're measuring the same thing, and we have also tried to relate those to the fundamental properties of the lung, namely, the bellows, action, the size of the lungs, the distribution of gas and gas transfer and we believe, through this method which can be got out, is an interesting one in doing two things: One, in enabling people to formulate a logical battery of physiological tests because you obviously don't need to use two tests which are measuring the same things; secondly, it does show you in relation to the X-ray and age, the type of physiological lesion which one gets in coal workers' pneumoconiosis.

(Applause).

BY DOCTOR SANDER:

Thank you very much, Doctor. I'm going to ask

Doctor George Wright to open the discussion. Will you come up, George, please? Doctor George Wright of the Trudeau Foundation who has done all the lung function work here at Trudeau. You going home?

BY DOCTOR WRIGHT:

Yeah, I was just on my way home.

It was a pleasure for me to say a few words about these last two papers. This is, of course, a field that I am intensely interested in and I suppose some of you had a bit of difficulty in following the enormous amount of data that was presented. I must confess to having had the same difficulty.

This sort of work does not lend itself well to easy digestion, and takes sometime going over in order to come up with an intelligent discussion. I shall confine my remarks, therefore, to some generalities.

First of all, most of the questions that I had posed for Doctor Motley have been posed for me by Doctor Jones. I believe that conditions here are a little bit different than in England. I can only envy Doctor Jones' opportunities to make a study of a group of individuals which embraces all types of experiences, in which he can set up his experimental approach and before he begins to get his men.

In our country, we're pretty much forced to take

the cases as they come, and I think that Doctor Motley would be the first to say that his material is not necessarily representative of the industry as a whole, but so far, we have not been permitted to go into a field and take all of the men or sample them in a random fashion, and I think perhaps the finest thing about Doctor Jones' presentation is that that is precisely what he has done. I wish that we had an opportunity to do that here.

I have some little doubt about this term breathlessness. All of us are seeking for some final criterion of a man's ability to work. Breathlessness is not a simple thing. It is an extremely complicated one. Those who practice medicine know that. The psychological aspects of exertional dyspnea are enormous, and I wonder whether or not breathlessness is an adequate criterion of a man's ability to work. It is certainly one of the things, but it should be subjected to the same scrutiny that Doctor Jones has subjected his X-rays and other tests to.

Now, at the end of this day, now, I had hoped that I would learn whether or not the coal problem in England was the same as that in the United States. Now, I must confess that I don't know whether it's the same. Now, I've had no experience with coal miners' disabilities, but it would appear to me from the paper of Doctor Motley and also Doctor Jones, that the disabilities are essentially the same

as that that has already been found and well described by McCann's group, by ourselves, and by others abroad, in silicosis that occurs in metal miners or foundry workers, in that the discreet nodulation and the linear exaggeration type of abnormality, little in the way of disability is disclosed, and that it's only in the conglomerate disease that one meets moderate to severe grades of disability. If I'm wrong in this, I would welcome correction.

I had wondered whether or not the changes in the coal mining industry were distinctly different from that in other industries. I still don't know, however, from the papers given by our American coal workers, as to whether or not what they are seeing in the coal industry is the same thing that is being seen by Doctor Fletcher's group in Britain.

I want to make one thing very clear. I know full well the enormous amount of work that has gone into these two papers, and I think the two men are to be congratulated on the very fine piece of work. I'm sure that as the years go by, we - they will be deriving more and more good information from the data that they already possess.

(Applause).

BY DOCTOR SANDER:

Do I hear any other comments from the floor or any questions? I'm sure you all have questions but you probably

don't want to ask them. No questions?

(No response).

Doctor Vorwald has a few announcements to make.
Thank you very much for the excellent papers.

BY DOCTOR VORWALD:

Members of the Symposium, during the course of the stay, you've heard some outstanding studies. I merely want to say many is the man who has pointed to the fact that we need more studies. I plead with you to make it available, make those studies available to us here, elsewhere in this country, whereby we might come further, come closer to the solution of the problems confronting us with respect to coal miners, coal workers and the pulmonary changes which they show. I think You will join with me in complimenting the speakers of today - Doctor Motley, Doctor Fletcher, Doctor Jones, Doctor Hugh-Jones, for the contribution which they have made to this Symposium. Thank you.

BY DOCTOR SANDER:

I'm very sorry I didn't give Doctor Motley and Doctor Jones an opportunity to answer the questions which were asked. Will you take over for a minute, Doctor Motley?

BY DOCTOR MOTLEY:

With regard to Doctor Jones' question on emphysema being a reduction in vital capacity only, that is not our experience. We find that the vital capacity is reduced,

but we find also that the absolute residual volume is increased. In our fourth group, the ones for advanced impairment, it's more than a hundred percent increased, that is the predicted residual volume is thirteen hundred; it's over twenty-six. In some instances, we have residual volumes as high as forty-five hundred c.c.'s. True, the vital capacity may be only two thousand, but we have the absolute increase in residual volume.

I did perhaps not make it clear in my evaluation of emphysema, we have really three things, the residual nitrogen percent in the expired air at the end, after breathing oxygen for seven minutes - if that is elevated, that is significant; secondly, the absolute volume as a residual volume with respect to the normal predicted, and thirdly, the residual predicted on volume.

I hoped - I tried to make it clear, I mean to point out the correlation with that arbitrary classification. When the residual volume is more than thirty-five percent of total on volume, lung volume, we have a fairly nice correlation with direct blood studies, getting rid of the carbon dioxide and lower, of the air in the air sacs as a result of the increased volume of residual air.

The ventilation factor, Doctor Jones, is simply a help before I analyze all my cases with respect to this figure. I used to evaluate independently, the residual,

the maximum breathing capacity. This simply gives me a figure. Now, by and large, if that is from eighty to a hundred, that includes a slight group, from eighty to sixty are moderate, sixty to forty an advanced and below forty far advanced.

Now, the only thing that upsets that, if the oxygen saturation rocks, and I think it is most important that we have blood saturations, because as I pointed out, we have normal resting saturations that may go down ten percent or more with exercise. That indicates disability. If that man is trying to work, he is working with a reduced oxygen saturation, a temporary degree of impotency, which we know increases arterial blood pressure, increases pulmonary vascular resistance.

Doctor Wright's question regards a conglomeration for total disability. In general, we find that is true. However, we do have some cases that would fall in Stage 1 or 2 or even in some, you would call it, normal X-ray, with a history of thirty-five or forty or more inside exposure, are totally disabled. We have found just as severe emphysema, Doctor Jones, in our coal miners as we have found in idiopathic emphysema in non-coal miner groups; some of them have gone up as high as seventy-five percent.

Doctor Jones, I would like to ask you why you use air consumption, as I understand that, that is the total

volume of air rather than the actual oxygen uptake on the basis of body surface area. Isn't there a possibility of air in there due to individual ventilating alveolae that are not profused insofar as arterial punctures go? We don't have any more hesitation in doing them than we do a venal puncture; it's just about as easy and we do that routinely. I have no trouble with our coal miner group.

It is true that we're not able to go out and take the whole coal mine and work it, that is these men have made application. Quite a number of the men that I have studied are still working. They made application to come in for study during their vacation period. We probably have, oh, around a hundred in such a category.

BY DOCTOR SANDER:

Doctor Hugh-Jones, do you have anything to close the discussion?

BY DOCTOR HUGH-JONES:

I probably have said enough, but there are just a few points that do occur to me. First of all, I don't want to sort of take up purely technical physiological points, but I think, Doctor Motley, I might - I must disagree with you again on one point, that you say that if a man's oxygen uptake and his carbon dioxide output are normal, then he's normal as far as his lungs are concerned. I don't think I quite agree with you there.

BY DOCTOR MOTLEY:

Just one part.

BY DOCTOR HUGH-JONES:

It seems to me it rather hinges on Doctor Wright's point about breathlessness and it also raises the last question Doctor Motley asked, why I measure air consumption. Breathlessness itself is a symptom and we were at pains to get somewhat - some way of consultatively measuring breathlessness and it is, after all, we think, the amount of bellows or bellowing and air shifting, if you like, that a man has to do, compared with the amount that he conceivably can do, which gives some measurement of breathlessness or what his sensation of breathlessness is.

I agree with Doctor Motley from the physiological point of view, we are all often interested, for other reasons, in knowing oxygen consumption, and also knowing carbon monoxide uptake which we often remember. The reason for carbon monoxide, there is no way known yet of getting over the back tension in the different parts of the lungs, in evaluating oxygen transfer, but there is the cardinal point which comes out from all this, and that is what is the cause of the breathlessness of pneumoconiosis, and I hope I made it clear that as far as we were concerned, we think it is exertional dyspnea, due mainly to the bellows not being able to have the reserve that they normally should have,

the maximum ventilatory capacity is reduced.

In a few cases, the air requirement, or if you like, the oxygen requirement, but the two don't run quite hand in hand, is increased, but that is subservient to the main cause, which is this diminution of bellows power in the lungs.

Now, the cause of that, and I think Doctor Vorwald this morning posed what were the causes of breathlessness in pneumoconiosis, and he suggested four, I think, and one was focal emphysema, another one was blocking of the arteries, another one was a ventilation profusion upset, and quite honestly, I think I would like to say that in the main, it's due to a fifth one, which he didn't measure or mention, and that is, I believe myself, that it is mainly a mechanical upset in the lungs, but we don't yet know the answer to this.

The causes of diminution of maximum voluntary ventilation is a thing which badly needs studying. Otis, in America, started a very useful method of thinking about it, and he points out that you can get diminution in the maximum bellows due either to the power of the muscles working the bellows, due to the diminution of the air flow, forms of bronchial spasm, due to a lowering of the vital capacity, which is the stroke of the bellows, or finally, due to the elastic properties of the lungs, the degree of

drag that you get through the actual shifting in and out of the lung substance, and we have not yet got down to that form of analysis, but it does seem fairly clear that in pneumoconiosis, the main effect is an actual reduced size of bellows which one gets and sees in the drop of the vital capacity, and it is interesting in a way, how well vital capacity comes out as a functional test in pneumoconiosis for that reason. I think rather surprisingly well, because it isn't really a dynamic test of function, as you will see from that model.

Then, lastly, there is this next question of what is the role of emphysema. Our results are, and if you will accept them, that's only if you will accept, that bad gas distribution could be defined as one of the effects of emphysema. Our results do suggest that the focal emphysema described by Gough, is not itself, of necessity, disabling, insofar as we have got, but that the same process, physiological process which gives rise to it, also upsets the elasticity of the lung substance, which diminishes the maximum breathing capacity, but that is speculation.

The whole business of what is emphysema, is an extremely difficult project, or idea, and we are trying to get down to analyzing how one could define physiological emphysema and how that overlaps with what the clinicians talk about and what the radiologists talk about, because

it does seem to me that the word 'emphysema' is shamefully misused in the sense that it is probably a syndrome which may give rise or may arise from various causes within the lungs.

And lastly, there is one point and that is about disability, which I hope I made clear. I did say that I thought conglomerate pneumoconiosis or complicated pneumoconiosis is the main and essentially really disabling part of the disease, but I think you will notice that in the simple disease, as age increased, and by the time people have got Category 3 simple pneumoconiosis at age fifty-five, they were beginning to get fairly disabled.

What happens after fifty-five we don't know. We didn't go above that, and I would stress that our results are limited over those years, because we didn't go beyond age fifty-five, which was the range we were interested in ourselves.

Thank you.

BY DOCTOR SANDER:

Did this discussion formulate any questions?

(No response).

If not, I want to thank all the speakers this afternoon, Doctor Friedman, Doctor Motley and Doctor Hugh-Jones, for this excellent discussion. Thank you very much.

(Session adjourned at 5:45 P. M.)

VORWALD COLL
BOX 89-91

SEVENTH SARANAC SYMPOSIUM

THE SARANAC LABORATORY of the
Edward L. Trudeau Foundation,
Saranac Lake, New York

Wednesday
September 24, 1952

PNEUMOCONIOSIS AND PULMONARY CANDER

Chairman: C. P. Rhoads, M. D.

Philosophy of Biostatistics as Applied to
Environmental Pulmonary Cancer

Morton L. Levin, M. D.

Discussion

General Review

W. C. Hueper, M. D.

Discussion

Asbestos Miners

Paul Cartier, M. D.

Discussion

Asbestos Weavers

Kenneth M. Lynch, M. D.

Discussion, led by E. R. A. Merewether, M. D.

BY DOCTOR VORWALD:

Ladies and gentlemen, it's past time to begin. We're behind schedule by twenty minutes. We hope that we will proceed in making up this time during the course of the day. I will admit it's a very tight schedule, but with the leadership of our chairman, I am sure that we will accomplish our objective for today.

Before we begin formally, there are just, again, a few announcements which I must make. I call your attention to the banquet tickets for tomorrow and also the

gathering at the Hotel Saranac tonight on colored photography, by the Ansco Laboratories.

In addition, there is a book here which was left on the seat. It is a brown book full of notes. I should like to read the notes, because I think I might glean in there some of the weaknesses of our Symposium, but nevertheless, here is the book. It has no name and I'm hoping that whoever owns it might, might get it here, so I'll put it right on the table.

With that then, we open our today's sessions on pulmonary cancer, and pneumoconiosis, from the inhalation of dust, and I think it follows logically, this topic follows logically along the same lines as we were discussing yesterday.

The Chairman for today's sessions is Doctor Rhoads, whom you know, and Doctor Rhoads is the Director of the Memorial Center for Cancer and Allied Diseases in New York City, and Director also of the Sloan-Kettering Institute for Research Study of Cancer. With that, Doctor Rhoads.

BY DOCTOR RHOADS:

Doctor Vorwald and Guests: I appear before you in a distinctly singular position. I have not been identified with the field of pulmonary cancer, nor with exposure to dust inhalation. I am in an always very difficult and

BY DOCTOR RHOADES:

Doctor Verwald and Guests: I appear before you in a distinctly singular position. I have not been identified with the field of pulmonary cancer, nor with exposure to dust inhalation. I am in an always very difficult and

much publicized position of research director, that unhappy type of individual trying to persuade or threaten individuals to do what he thinks they ought to do. He always finds this very difficult indeed, of course, if not impossible.

However, I was very happy to be able to accept Doctor Vorwald's invitation, because in our group and other groups associated with us, have come certain aspects of work which pertain, I think, to your interest and to the field of pulmonary cancer. I have no comments in the field of statistics, and so will leave that important topic to those who will present papers.

I was impressed, however, with the program, to observe what seems to be a strongly inclined point of view toward the purely industrial hazard aspect of this problem and perhaps less attention given to diagnostic, therapeutic and experimental considerations, which perhaps play an interesting role.

Until we can, through your efforts, eliminate the cause of this very serious condition, we must consider what can be done better to handle it once it has occurred.

We have been interested, in our institution, in the development of techniques. We permit study of therapeutic problems relating to pulmonary and other environmentally induced cancer. We are very happy, some years ago, to be able to provide facilities for Doctor William Smith,

which have permitted the development of highly precise and satisfactory techniques for carcinogenic principals.

Many of us have felt that that enterprise subsequently conducted at New York University, in the Medical Department, has provided important data, indeed principals which may lend themselves to study of pulmonary as well as other environmental types of cancers. I refer, of course, to the study of that form of cancer induced in experimental animals and conceivably in man, by the positive action of high temperature catalytic agents, fuel oil.

Doctor Smith and his associates were able to demonstrate the cause of these activities of these compounds of this material, and of certain compounds contained therein. There are strong suggestions that no one pure compound induces the neoplastic change, but rather a combination of several. There was also strong evidence that this combination of materials will induce cancer in the primate, which I regard as an important advance.

If we have, then, the principal of possible multiple origin or whole carcinogenesis, that provides at least leads for further investigation. We have also been happy to have with us Doctor Winter who came subsequent to his major observations with Doctor Ames in St. Louis. I am a strong defender of Doctor Winter, whom I regard as an energetic and reliable worker. I am aware that his statistica

has - have been brought into question, but his tables, his data, with those of others, would seem to many of us to provide adequate evidence to suspect the inhalation of tobacco smoke from cigarettes as a participating etiological factor in pulmonary neoplastic disease. Doctor Winter has never, as far as I know, claimed that this was the only factor, but only that it is an important one which justifies further study. We have been fortunate to have certain observations which we think may give techniques suggesting the possibility of further study, both of etiologic, of a prophylactic and a therapeutic nature.

May I have the first slide, please? I am going to show just a few slides which indicate certain areas of study, and of thought which may amplify the topics of today's discussion. I presume that you have all seen the article of Stott's which appeared in the summer issue of the Bray's Journal of Cancer. If you have not, for your interest, I throw it before you, because it does amplify those studies of the individuals who have been concerned with smoking as etiologic and pulmonary canceration.

This is the chart of the London areas and suggests very strongly a concentration of pulmonary cancers in those areas heavily contaminated with industrial smoke and a trend toward the areas to which the prevailing wind blows. This may, in the opinion of Stotts, which seems to be well

established by the evidence, provide an additional factor to the one referred to by those concerned with cigarette smoking. And I would like to emphasize again the possibility of multiple factors in the etiology of pulmonary neoplastic change.

Doctor Winter has allowed me, since he is in Europe, to present one example of cancer in the experimental animal, induced with the production of the burning of cigarettes under circumstances which are considered to be related or reasonably close to those which obtain in man. True cancer now has been induced by these pellets and it is hoped that it will enable us to go forward to a more exact knowledge of the physical circumstances involved in smoking, to be developed, and more adequate evidence concerning the nature of the active principal and the mode of its production and the mode of preventing the lesions thereby caused, can be had, but this is a true cancer in the experimental animal, caused by cigarette smoked tar.

Now, we have an important area in the early diagnosis, and I hope that those concerned with public health measures, Doctor Levin and others, will refer to the question of how well we can pick up treatable disease by mass survey. In our own experience of about one hundred fifty to two hundred patients a year, with pulmonary patients, we find that the pick-up that comes to us from that survey is

comparatively limited, but there is a very active pick-up from armed service examinations and from examinations in the doctors' offices or the hospital.

This suggests some practices in bringing before the practitioner, the importance of considering neoplastic change as etiologic, as possibly suspicious of any pulmonary lesion, and I do hope we will learn more of early diagnosis by means of mass survey techniques.

Next slide. Of course, in our particular interest, we are deeply concerned with getting a symptomatic disease in a stage where it's still treatable and we're very happy to be able to report that steadily the incidence of operable disease increases, in part because of the greater competence of the surgeon and to some extent perhaps, because of the early rendition because of adequate diagnostic measures, and these are very important factors from the standpoint of the life of the effected individual.

So, we are now working on some thirty percent of the patients who present pulmonary lesions. Now, very happily, we have been able to define - to devise and apply a more radical technique of surgical removal. For many years, it was regarded as impossible to attack the lesions involved, and, of course, it is their involvement which renders the pure rate so low in pulmonary cancer. Daszda, Hand, Watson and associates, have devised a procedure now

in general use, which includes in the red line, accessible mediastinal nodes and some fifty patients have been subjected to this radical dissection, applying the principal, the same principal of bi-section which has been so successful in X-ray surgical patients in cancer.

And, we are able to report at this time, some suggestion of to what extent this may be an advance. It's not a very emphatic suggestion, and we have no idea what will be the factor at the end of the five-year period, but we are satisfied, as testified by the slide, that once the disease had gone beyond the nodes directly contiguous to pulmonary tissue, it is hardly worthwhile to carry out these elaborate bisections.

On the other hand, we are satisfied that there is good reason, at least, at present, to attempt to remove directly contiguous involved nodes, because we do have these four individuals who, otherwise, would have been fatalities, who may well have been benefitted by this rather radical operative process.

Next slide, please. I have been very much concerned and my associate, Doctor Southam, in the whole question of potential hemotherapy of pulmonary cancer. As Doctor Margus remarked to me, it is conventional to say it is cancer and nothing to be done. Of course, it is true that it is not treatable, the patient will not be cured, but

when a man comes to you desperate with dyspnea, deeply cyanotic, because of superior mediastinal pressure from his lung cancer, he is gratified and thankful if you can provide him even a few months or weeks of relief. This can be done in many instances today, with those conventional therapeutical - hemotherapeutical agents of the tri-millimeter service, which you are all familiar with, and I simply throw on these slides to indicate the evidence that there is a transient therapeutic result to be obtained and this should not be sneered at.

In one sense, it indicates that with careful care, we can achieve better therapeutic results, and secondly, the possibility of relieving a desperate situation for weeks or months, should be considered.

We have all been seeking methods by which we could extend the efficiency of hemotherapeutic attack. This has involved some procedure which would allow reproducible experiments on human pulmonary cancer. In our laboratory, Doctor Toolan has been successful in drawing epidermoid cancer of man of a variety and type, in the experimental technique, and applying this technique employing essentially successful hemotherapeutic methods.

This (slide) is a low power of a bronchogenic from man as removed in the operating room. This (slide) is a high power -- I'm thinking of no professional diagnosis,

-- and here, after two weeks of experimental animal, low power, and the next slide, the same preparation high power, two weeks in the animal. This can be done quite regularly and lends itself candidly to various factors and techniques.

Now, this is an example of how epidermoid cancer, this is not a lung - in the experimental animal, cancer planted in the radiograph, can be destroyed completely either by hemotherapeutic or viral means. This is a destroyed implant of human epidermoid cancer.

Similarly, we are making great efforts to grow human pulmonary and other forms of epidermoid disease in tissue culture. It can be done not as well as we would like and this is an example. This is epidermoid disease, but not pulmonary, growing in tissue culture at the end of a ten-day period.

And this (slide) is - this is the treated culture, the first, the one before the control, infected with a virus suitably adapted to the destruction of certain forms of human cancer under artificial conditions, and you will observe that the cancer cells have been destroyed leaving the normal cells. This can not be done in every form of human cancer, but it can be done with a good many and we hope that they will come to light through the development of these techniques, with better diagnostic, better therapeutic

methods and then we will wait for you gentlemen to give us prevention of the disease entirely.

Now, the first paper is Doctor Levin's. You're all familiar with his great contribution to this field, and I personally have waited with great interest, his analysis of the data which bear on the environmental pulmonary cancer. Doctor Levin.

BY DOCTOR LEVIN:

Thank you, Doctor Rhoads. Ladies and gentlemen, I am supposed to talk to you on the philosophy of the biostatistics of environmental pulmonary cancer, and I have never been sure, up until this moment, whether I was supposed to concentrate on philosophy or biostatistics or environmental pulmonary cancer. I shall probably try to do all three, and succeed in neither of them or not one.

I certainly would like very much to second the remarks of Doctor Rhoads as made and perhaps I can do so and make some comments on some of the things he has said, by telling you what my own point of view is in this matter.

It is primarily that of an epidemiologist which is the field in which I received most of my medical training. An epidemiologist, as you probably know, is not a statistician necessarily, although he has to know something about statistics and use them and know when he is in trouble, some - so he can go to someone who is a statistician.

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An epidemiologist is, primarily, a fellow who is interested in two questions, first of all, whether a certain group of people actually do or do not have more of a certain disease than other groups do have. He's interested in knowing whether that's really so, and so he is rather critical of things which may look like that, and nevertheless are not really good evidence, and he is interested in knowing, if that is so, what the reasons might be, and his reasoning for those interests are quite practical, because he is the man who is usually called upon to make certain decisions or to give certain advice as to whether certain things should or should not be done in a given situation.

Now, it just happens that historically, that type of question usually arose with our epidemic diseases and that's why, I suppose, the term epidemiology, is usually thought of as referred to with people who deal with infectious diseases, but precisely the same question can arise in almost any type of disease, and certainly they do arise in industry.

Now, the epidemiologist's point of view on pulmonary cancer and the evidence respecting various etiological agents is, I would say, highly critical. That is, we are aware from long experience, that a lot of things which look like etiological relationships are not necessarily such. The other point of view, I would say, perhaps

would seem to you to be opposite, but I would offer to you as the result of long reflection on the part of a good many people.

We epidemiologists suspect that, from the standpoint of the causation of pulmonary cancer, our best experimental animal may be man rather than any animal we can study in a laboratory. Now, man is not a very good experimental animal, but it seems as if in cancer, he may be our only one in some forms, and the laboratory may or may not confirm what we find in man, but we have a strong feeling that we are missing a pretty good bet in not studying much more intensively, possible relationships which may be etiological to man.

We have about twenty-five thousand cases of pulmonary cancer each year in this country, and it seems very unlikely that those twenty-five thousand people do not have a good many characteristics which distinguish them from their fellows, which may have some relationship to the fact it involves lung cancer, about which we still do not know very much.

Now, Doctor Rhoads mentioned the relationship to smoking tobacco, especially cigarettes. I know of no observation which would imply or implicate any other type of smoking, and I mentioned the work of one investigator or rather two, Winter and Graham. Now, I happen to know Doctor

Winter and I like him very much, and I agree with Doctor Rhoads' estimate of him as a very energetic, a capable and an enthusiastic chap. The kind of observation, however, which Winter and Graham made about relationship between smoking and cancer is precisely the kind of observation which you will find in the literature for the last twenty years. If you look up one of the earliest multigraphs on lung cancer published about 1930, you will find about four pages devoted to the relationship to smoking cigarettes.

Now, those observations were never taken seriously, and they were never picked up, precisely because of the uncritical, over-enthusiastic type of analysis on which they were based, in other words, they were based on inadequate controls, they were studied in such a way that one could see that the person making the study has a pretty strong bias in the first place, as to what he would find. It's not the sort of evidence which really tells you anything that you can rely on.

Now, it happens that the observation of Winter and Graham have been confirmed as to their general tenor. There is a relationship between smoking in tobacco. There is a great difference in the quantitative observations. If we are to believe the observations of Winter and Graham, pulmonary cancer is extremely rare in people who do not smoke cigarettes, about three percent of lung cancers,

according to their data, three to five percent, occur in people who do not smoke cigarettes.

That is not in accordance with observations which we have made, and I, frankly, doubt whether it is in accord with what the facts are. I think that is one of the crucial questions which remains to be proven or to be further tested.

However, I did not intend to really go into the question of that particular observation, because that is not our topic. What I thought I would do is to give you some general quantitative background for this whole question of lung cancer which would serve as sort of a backdrop for the papers which are to come, first of all, the importance of this topic, this form of cancer as a human tumor.

May I have the first slide? This first slide is simply the characteristic age curve of cancer, that is the incidence of cancer by age among males and among females as usually occurs in New York State where, as you know, cancer is a reportable disease. These are all cases reported from all sources for various periods of time, put on an annual basis. That is a very interesting curve, for one thing, because it does not follow the type of curve that you would expect in most forms of cancer which keeps going up with age. Most forms of cancer start in lower, younger age groups and keep going straight up. In lung cancer,

after about fifty-five, the rate drops.

There are a number of things which can be said about that observation, and I don't propose to go into all of the possible explanations, but it does mean, one thing, it does suggest one thing, that for some reason, people who were born, this is 1945 roughly, and people aged sixty in 1945, were born in 1885, as my arithmetic informs me. People born before 1885, for some reason, had less, have less lung cancer than people born at an earlier age, an at earlier period.

That may not be self-evident to you, but it follows from a further analysis of these curves which show that when you analyze these curves by the year of birth, they keep on going straight up, and they keep on going up in a much higher fashion depending on when the person was born. That is not the sort of thing that you would expect in a carcinogen, if it is a carcinogen, which occurs generally, such as a carcinogen in the air. It's apparently something which picks out certain ages and not others.

Now, in most - the important point that I wish to bring out in this slide is that you couldn't analyze any two groups of people, say the number of lung cancers in two factories, or the number of lung cancers in one factory as compared with the number of lung cancers in the surrounding countryside, unless you took into account the age factor,

because obviously if you did have a lot of people in this age group, you would have more or less cancer than if you had a lot of people in this age group or this age group. You would certainly have to adjust for age, and it's surprising to find that some studies do not even take into account such a simple and obvious variable which has got to be adjusted.

Now, when you take this type of curve, that is the number of cases which occur by age in one year and apply to an ordinary life table, which is the expectancy, the expectancy of living, at various ages in the duration of life, you will get a table such as the next slide, which will tell you about how many cases of cancer of various types one might expect to occur in the population today throughout the rest of their lives. These figures are all based on an a base of one, that is this top figure gives you the probability of skin cancer occurring in males throughout life, and the figure .02 means that two out of a hundred, two percent of all males, almost two and a half percent of all males, using the type of data which I have just described, that is the actual incidence of cancer as determined by reporting applied to the life table, two out of a hundred males throughout the rest of their lives will develop some form of skin cancer.

Now, for lung, the figure is just over one percent,

about one percent of all males according to New York State data, may be expected to develop lung cancer. That may be considered a sort of backdrop of this whole problem.

It is a problem which, at a minimum, will effect one percent of the male population. I'd like to stress the point that that is a minimum. First of all, these figures are based on the data for '42, '44, since then, the rates for lung cancer have gone up and the death rates from all causes gone down, which means that the opportunity for developing lung cancer has increased and the rate at which it develops has also increased, so that figure one percent is definitely minimum, but it is at least one percent of the general population.

May I have the next slide now? This -- oh, there is a paper slide in between there -- may I have the first of the paper slides then? I don't know whether you can see this. I think this is another of the important observations about lung cancer which we have to remember concerning this whole problem, and that is the increase which has occurred over the past two or three decades.

These are the actual mortality rates in New York State between 1931 and 1948 in females and in males, standardized for age, that is the age factor has been kept constant in the two periods. The striking thing here is the difference in the increase in the sexes. In females, the

increase has been about from 2.5 to 4.3 deaths per hundred thousand per year, an increase of about seventy percent. In males, the increase has been from 4.7 to 19 per hundred thousand, an increase of over three hundred percent.

Now, an increase in lung - in any form of cancer, as you know, may be due and often is attributed to the fact that our diagnostic procedures are more effective. If that is the explanation for this increase, then we would have to ask ourselves why, diagnostic procedures were three hundred percent more effective now as compared with 1931 in males, but only seventy percent more effective than in females.

I would say that certainly that causes or casts considerable doubt on the explanation that such an increase can be due to diagnostic procedures alone and makes it extremely probable that there has been a real increase in lung cancer over this period, probably in both sexes.

Another bit of evidence -- may I have the next slide -- which leads in the same direction, is the different way in which the ages have been effected in this increase. Now, that's the slide that you showed, the last slide that you showed before this one. That's it. I - this is the mortality rate in the United States. I call your attention to the upper left, your upper left-hand corner here which gives you the age specific mortality rates.

This is age down here in '30, '39 and '49, and you will notice that the increase has been more marked here in the middle period than here in the older periods, which again coincides with the observation that there is something happening in these people around sixty, which apparently is not happening nearly so markedly in people who are older than that. That again is the sort of thing that one would not expect to see if this were purely a diagnostic phenomenon. There is no reason to suppose that diagnosis may - in a disease with high case fatality such as cancer of the lungs, should make such a marked difference according to the age, should show such a marked difference according to age alone, so that putting these data together, one would lead to the conclusion that one could not safely assume that this increase was purely artificial and due to the fact that we found more lung cancer, one would certainly have to assume that there was a very strong probability that this was something real.

Now, one of the points that one can make about real changes in cancer incidence over time, is, as a matter of fact, in incidence in any disease over time, that if they are real, they are probably due to some environmental factor. The reason for that goes back to one of the laws of genetics, if you will remember your genetics. You will recall that, according to the way the genes operate, their

proportion from one generation to another remains constant under most circumstances, so that if genetic factors, if constitutional factors were the ones which obtained, we would not expect the incidence of the disease to change from year to year. If we find a real change, we should suspect some environmental cause rather than some genetic cause as the reason for that change.

So I would feel safe, from the philosophic viewpoint here, speaking as an epidemiologist that we have good reason to suppose, first, that there has been a real change in lung cancer and, second, that that change is probably due to something in the environment. Whatever that may be, of course, remains to be seen.

Now, how would we go about studying the possible relationship of lung cancer to various environmental factors? Now, may I have the next slide. I have placed on this next slide, a hypothetical situation in a - I don't know whether you can see this or not. I have here a state or a population group in which there are five million males in whom nine hundred cases of lung cancer occur in a year, and we have in that same population group an industry, we call Industry A, which has ten thousand males among whom nine cases of lung cancer occur in a year, so that in the entire group, that is in the entire state, if this is a state we're talking about, there are nine hundred and nine

cases of lung cancer in one year.

Now, suppose one wanted to study lung cancer as to whether it was related to, say, some particular industry. According to -- we could use at least two methods which might be called the case method or the retrospective incidence method. We could take all nine hundred and nine cases of lung cancer, as we do here, and we could take comparable number of controls, we take a thousand controls, and if we were interested in Industry A, if we had reason to suspect Industry A, we'd find out how many of these nine hundred and nine lung cancer cases were people who worked in Industry A. If we got all our cases, we'd find that there were nine of them and in our control group, which should be representative of the total male population, we should find the same proportion of industry A workers as ten thousand is to five million, that is the proportion of male population to males in industry A.

Now, ten thousand to five million is two per thousand, so we'd find two cases, two people who worked in Industry A in our controls. Now, mind you, I assume that we don't know these facts. We simply have these nine hundred and nine cases and we're interested in whether Industry A has anything to do with lung cancer. If that is what we did, and we were successful in getting all nine hundred and nine lung cancer cases and if your control group is

properly selected, in other words, it wasn't biased in such a way as to accidentally drop out these two Industry A cases, or if our lung cancer cases were poorly selected so we might not - we may have missed these nine cases, in other words, if our experiment were perfect, we'd find that there was one percent Industry A people in the lung cancer cases and two per thousand Industry A people in the controls, which amounts to a ratio of five to one, that is one percent is five times as much as two tenths of one percent. This gives you five to one.

Now, if you go back to our postulated population, this is a method, nine hundred and five million is a rate of ninety per hundred thousand and nine per ten thousand is a rate of eighteen, it's just - it's just the other way around. This is eighteen per hundred thousand and this nine should be here and the relation between eighteen and ninety is five to one, which is exactly what you would find in your case method.

In other words, your case method, if it were properly performed would give you the true situation with respect to the incidence of lung cancer in industry A as compared to the population. If there was five times as much lung cancer in Industry A as in the population, assuming, of course, that the age factor and other possible variables which might effect lung cancer had been given

consideration, I am assuming that that has been done. The other way in which you might do that would be the retrospective incidence method. You take ten years of Industry A and how many cases of lung cancer occurred in ten years.

Now, if nine cases occurred in one year; in ten years, ninety cases could be expected to occur in the ten years, and they'd be - you'd have to ask yourself, how many cases do occur in that period, and one of the ways in which you might answer that question is, why, you would say, I expect or would expect the same number of cases that would occur if industry A had the same incidence as the general male population. That would give you ten years times ten thousand males, which is the same thing as a hundred thousand and one, and an incidence of eighteen per hundred thousand would give you an expected case incidence of eighteen cases, in this period, which again would be a ratio of five to one.

Now, I'd like to call your attention to the fact that if this particular case method were done on half the number of cases, if instead of getting all nine hundred and nine cases, you had gotten half of those, then these two observations would not be significantly different from each other from the standpoint of chance variations. That is they would be the kind of difference that would occur often by chance alone, so that one couldn't be sure that that was

a real difference, so that the actual magnitude, the actual number of cases that you'd study in an industry or that you'd study of this kind, is as important as the selection of the cases themselves.

Now, let us assume, however, that we have done this study in this way and we do find a significant difference, in other words, we find that there are five times as many cases in Industry A as in the general population, all factors being held constant. Does that mean necessarily that Industry A, there is something in Industry A which causes lung cancer?

Now, in my view that does not necessarily mean that. I think that one of the important principals, philosophical principals, to remember in studies of this kind is that data do not tell you any more than what they actually signify. They tell you that, for some reason, people in Industry A have developed five times as much lung cancer as people in the general population. That may be due to the fact that there is something in Industry A which causes lung cancer. It may be due to something else, for instance, if we were studying a factor such as the presence of six fingers on one hand rather than five, and found that one industry had five times as many people like that as would be expected in the general population, I think one would think twice before one attributed that to something in the

industry. One might, normally, if that is a genetic trait, one might wonder whether the owner of the industry had six fingers and whether he had many members of his family working for him.

The point is that one can not automatically jump from association of this kind to causation. Causation may be the factor or it may not. Whether or not it's causation depends on other evidence, depends on what else we know about the disease. If we did not know that various dusts can produce lung pathology, that it is possible to produce lung cancer in animals by the application of certain chemicals, I think we would be less apt to suspect that an observation like this actually indicated a cause of the effect.

Now, actually, there are only a few ways in man that I know of, whether - or that I can think of, in one connection, that can actually prove causation to one's satisfaction. One is by this second method here which I have called experimental induction. I mean if one suspected a certain substance in this industry and one applied it either to man, which, of course, is extremely improbably although it has been suggested, and as a matter of fact, we are making tentative plans along that direction, if one applied to man or animals and actually got the disease in question, I think one would have some additional evidence

that that was an etiological agent.

However, I'd like to call your attention to the fact that if one didn't do that, one wouldn't have evidence that it wasn't an etiological agent, because unfortunately, there are many things or at least there are some things which will cause cancer in man that will not cause it in animals, so the negative evidence is not conclusive negatively.

The other way you can test it is by the protective test, by actual prevention. I mean if we actually made some element in Industry A which was a factor and then studied the incidence later on, the protective incidence and found out it had dropped, I think we would have some additional evidence to the etiological relationship, but it seems to me that one or the other of those tests has got to be applied in all of our presumed data regarding etiologic - etiology in pulmonary cancer, environmental etiology, before we are absolutely certain that what we think is etiology is or actually is.

Now, I don't know whether I'm running too much over my time. I had some data which, from the philosophical point of view seemed to me to be interesting as to how important environmental, especially occupational cancer might be in America, but we might keep that, perhaps, for the discussion, Mr. Chairman.

BY DOCTOR RHOADS:

You have three minutes.

BY DOCTOR LEVIN:

I do have three minutes? Well, may I have the next slide? These are some - some of our data regarding the relationship between cigarette smoking and cancer, two forms of cancer, cancer of the lung and cancer of the larynx. This is simply the proportion of cases of cancer of the lung and cancer of the larynx who were cigarette smokers, by age, compared with two types of controls, in the same hospital population.

This is our - the Buffalo Hospital, the two controls were patients who had no malignant tumor and patients who have other types of cancer than lung or larynx. You will note one thing which apparently has not been emphasized very much, but which we have confirmed by further study of the same phenomenon. That is apparently there is much relationship between cigarette smoking and larynx cancer as between cigarette smoking and lung cancer.

You will notice, also, the interesting thing is that the cigarette smoker is one of the things which drops off with increasing age. You were caused - one of the things we would probably have to find to explain the age curve in cancer is something which dropped off with increasing age. For some reason, at least at present, the older

a man is, the less likely he is to be a cigarette smoker.

Now, that's a very interesting observation. That is, again, as Doctor Rhoads indicated, and I think we will all agree, this does not necessarily mean that cigarette smoking causes lung cancer, though that may be simply an added effect or factor. It simply means there is more lung cancer among cigarette smokers than there is among non-cigarette smokers.

The interesting question is what does that actually mean from the standpoint of numbers, quantitatively? And there are various ways of doing that, and I won't try to go into the arithmetic of it, but on the next slide, I have jotted down some conclusions which arise from our data. They do not arise, incidentally, from the data such as has been induced by Winter and Graham, because if their data is right, practically all - practically only cigarette smokers get lung cancer and that isn't true according to our data at all.

Thirty-five percent of lung cancer cases are not cigarette smokers according to our figures. If our data are correct, throughout life, this is the incidence of lung cancer. In non-smokers, five per thousand, this is throughout life, mind you. In heavy smokers, that is not just cigarette smokers, that's people who smoke one to two packs a day, it's six times as much or thirty per thousand, that's

three percent. You will recall that the lifetime expectancy in the general male population is one percent and this is three times as much.

Now, according to a recent paper in Public Health Reports by Britain, Frasier and Coven, it's is my opinion that that agrees at least in general with the data of Machle and Gregorius on chromate worker cancer; these are not the figures given by Britain, Frasier and Coven, they are simply my interpretation of them, as to how much lung cancer those workers may be expected to develop in twenty-five years if their data are correct, per thousand.

Among white males, there should be sixty-five cases in twenty-five years in chromate workers and among non-white, two hundred and thirty-five. Now, unless my arithmetic is very, very far off, this means one thing, possibly two, certainly that cigarette smoking, if it is a carcinogen is a pretty mild one, since it increases the general incidence throughout life only by a factor of three, and if such an occupational factor as chromate dust is a carcinogen, it apparently, when it does work, is a pretty strong one, since it may increase it by a very much higher factor. That is, twenty-three percent of all the male workers, in twenty-five years, may be expected to develop lung cancer if the data published by these workers stand up and are representative of the true picture.

So that we have apparently, assuming that the facts we have at our disposal, we can rely on, two chief types of carcinogen, which are environmental, among others, because I hadn't considered general environmental dust, one which is heavy smoking, which is apparently very weak, and one of this type whose incidence we don't know since we don't know all the occupational carcinogens, but which when it does occur, is perhaps very strong.

I think I have more - one more slide, which perhaps puts this in numbers. Applying the one percent throughout life figure to the seventy-five, approximately seventy-five million males in the United States, throughout their lifetime, seven hundred and fifty thousand of them will develop lung cancer. Of those, a little less than half or rather something like a third, two hundred twenty-five thousand, will be non-smokers. The rest will be smokers, but since there is no reason to suppose that smokers are immune to the same influences as the non-smokers, we can only attribute three hundred and thirty-seven thousand of those cases to smoking per se, so that what this leads us in substance, to is this, that even if smoking gets full credit, we have got to account for about four hundred thousand cases of lung cancer to other causes which, if they're environmental and if they occupational, means that we've got to find a lot more occupational causes of lung

cancer than we have up to the present.

The one thing which makes that likely is the fact that one, when occupational causes do occur, they apparently are quite strong as compared to such a mild agent as tobacco seems to be. (Applause).

BY DOCTOR RHOADS:

I know Doctor Levin's presentation will arouse a great deal of discussion. I would like to open the discussion by just one question, Mort. I assume that the drop-off with age is just for the number of individuals in that age group population?

BY DOCTOR LEVIN:

Yes, that's the case.

BY DOCTOR RHOADS:

And I also assume that when you refer to the high incidence in individuals born in 1885, that is continuing high as the years go on and similar studies are being made; it isn't that year, but that period after?

BY DOCTOR LEVIN:

Yes.

BY DOCTOR RHOADS:

I'm very much interested to hear Doctor Levin indicate his acceptance of smoking as a possible etiological or participating agent, because this, in view of the importance of the problem, its extent would justify one perhaps

in considering whether that subject should lend itself to experimental work designed to prove or disprove the case more adequately, provide more adequate statistics on the subject.

Now, may we have discussion? Doctor Lanza, would you care to make any comment at all?

BY DOCTOR LANZA:

No, I have no comment.

BY DOCTOR RHOADS:

Is there no one who inclined to smoke cigarettes, who wants to ask questions?

BY DOCTOR FLETCHER:

According to Doctor Levin, he supposes that chromate is a much more powerful carcinogen than tobacco, but how did he measure the intake of chromate as compared to tobacco in these two groups?

BY DOCTOR RHOADS:

Doctor Levin, would you care to answer that question of Doctor Fletcher's?

BY DOCTOR LEVIN:

Well, I think it's a very good question. In other words, it refers to the relative dosage. I wasn't trying to be as fine as that. I was simply referring to chromate as the net experience of a chromate worker, wherever they're involved. The net experience of a chromate worker, if the

data that we have available act or are correct, I have no reason to suppose they're not, because they seem to have been very carefully done. The net experience, whatever that is, is apparently much more powerful than the net experience of heavy smoking. Now, your question is what would happen if we took those chromate workers and gave them one tenth as much dust as they did get? I don't know. I'm simply referring to the actual facts as they were observed in actual life.

BY DOCTOR RHOADS:

Does that answer the question? I'm surprised no one has commented on the possibility of air pollution. Doctor Smith, you must have some feelings on this point. I saw Doctor Joe Smith in back here a few minutes ago. Would you care to comment on air pollution in view of Stott's report, as a complimentary factor of environmental nature?

BY DOCTOR SMITH:

I have yet to see Doctor Stott's report. It certainly opens up a very different problem. I think I'm in a fortunate position here in that most people who are really familiar with this subject and who told me what little I've been able to gather about it, are in this room and I can pass the problem on to them.

BY DOCTOR RHOADS:

Well, there are other facts bearing on this

question in one report, the incidence of lung cancer in individuals in cities of over one hundred thousand, is much greater than in rural communities. I don't know how valid these hints are, but they justify further discussion. Are there further comments on Doctor Levin's paper?

(No response).

If not, we'll proceed to the next paper. Doctor Hueper needs no introduction. You know his long continued contributions to the field of environmental cancer. My acquaintance with him dates over many years. It has always been most stimulating. He is one who has stuck to his guns in this important problem, and I happen to know, in some instances, under difficult circumstances, and has given us much knowledge of this important field. Doctor Hueper.

BY DOCTOR HUEPER:

(Doctor Hueper read a prepared paper, which is on file at the Saranac Laboratory).

BY DOCTOR RHOADS:

I'd like to congratulate both of the speakers on completing their presentations on time. I hope their precedents can be observed for the rest of the day.

I would like to raise one or two questions to initiate the further discussions. Doctor Hueper, you referred several times to the presence of benzpyrene in suspected material as supporting the view that these materials

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Occupational and Environmental Pulmonary Cancer

COPY

With Special Reference to Pneumoconiosis

W. C. Hueper

National Cancer Institute

National Institutes of Health

United States Public Health Service

Department of

Health, Education, and Welfare

Bethesda, Maryland

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OTIS HISTORICAL ARCHIVES
NATIONAL MUSEUM OF HEALTH AND MEDICINE
ARMED FORCES INSTITUTE OF PATHOLOGY

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"Do you realize that within the lifetime of men now living, within a hundred years or one-hundred and thirty years at most, all the external conditions under which man lives his life on this earth have been more completely revolutionized than during all the ages of recorded history which preceded."

R. A. Millikan.

Occupational and Environmental Pulmonary Cancer

With Special Reference to Pneumoconiosis

W. C. Hueper

1. Increase in Lung Cancer Frequency.

It is generally recognized that exposure to certain exogenous physical and chemical agents plays an important role in the causation of cancers of the skin. Considering the fact that the respiratory tract may be looked upon as an inverted part of the outer covering of the body and that many of the known or suspected cutaneous carcinogens also are inhaled, it is not surprising that observations made mainly during recent decades have brought to light a rapidly increasing number of environmental and occupational agents which are involved in the development of cancers of the nasal cavity, paranasal sinuses, larynx, and lung. The relative importance of these discoveries is greatly enhanced because of the consistent and spectacular increase in the frequency of cancer of the lung noted during the last 50 years in almost all industrialized countries (Hueper). This rise in the number of deaths from pulmonary cancers has reached in several regions such proportions, that cancer of the lung has replaced cancer of the stomach as the most frequent cause of death from cancer among males (Steiner, Butt and Edmondson; Halpert).

It has lately become a convenient expedient for some investigators to explain away unpleasant things in vital statistics, such as the rising death rate from bronchogenic cancer (Stocks) by changing fashions in certification of causes of deaths, increased awareness of the medical profession of lung cancer, progressive aging of the population and similar half and part truths. However, to the critical observer there exists little if any doubt that an appreciable part of this development is real. The following graphs (fig. 1 and fig. 2) and table (table 1) giving statistical data from this country and abroad support this statement.

Fig. 1. Death Rates for Lung and Larynx Cancer in the United States, 1925 - 1948.

Fig. 2. Lung Cancer Death Rates for England and Wales, 1900 - 1947.

These observations made on official death certificate data are in agreement with the evidence obtained by investigators using autopsy material, Steiner, Butt and Edmondson found at the Los Angeles General Hospital that carcinoma of the lung was noted during the period 1923 - 1927 in 0.6 percent of all autopsies and constituted 4.3 percent of all tumors, while in 1943 to 1946 corresponding figures were 2.3 percent and 11.3 percent. Similarly, Beeler and Iray observed at the Lotterman General Hospital in San Francisco bronchogenic carcinomas in 1.2 percent of all autopsies during the decade 1920 - 1929, whereas they occurred in 2.7 percent in the period 1940 - 1948.

Corresponding observations were recorded by Ochsner and DeBaakey; Rosahn; Gowan; Metropolitan Life Insurance Company, and other American investigators (Hueper; Steiner; Bruby and Sweany). The statistical data of Dorn and Potter show that this trend has been in existence since 1914 and has shown a tendency in late years to become less pronounced (table 1).

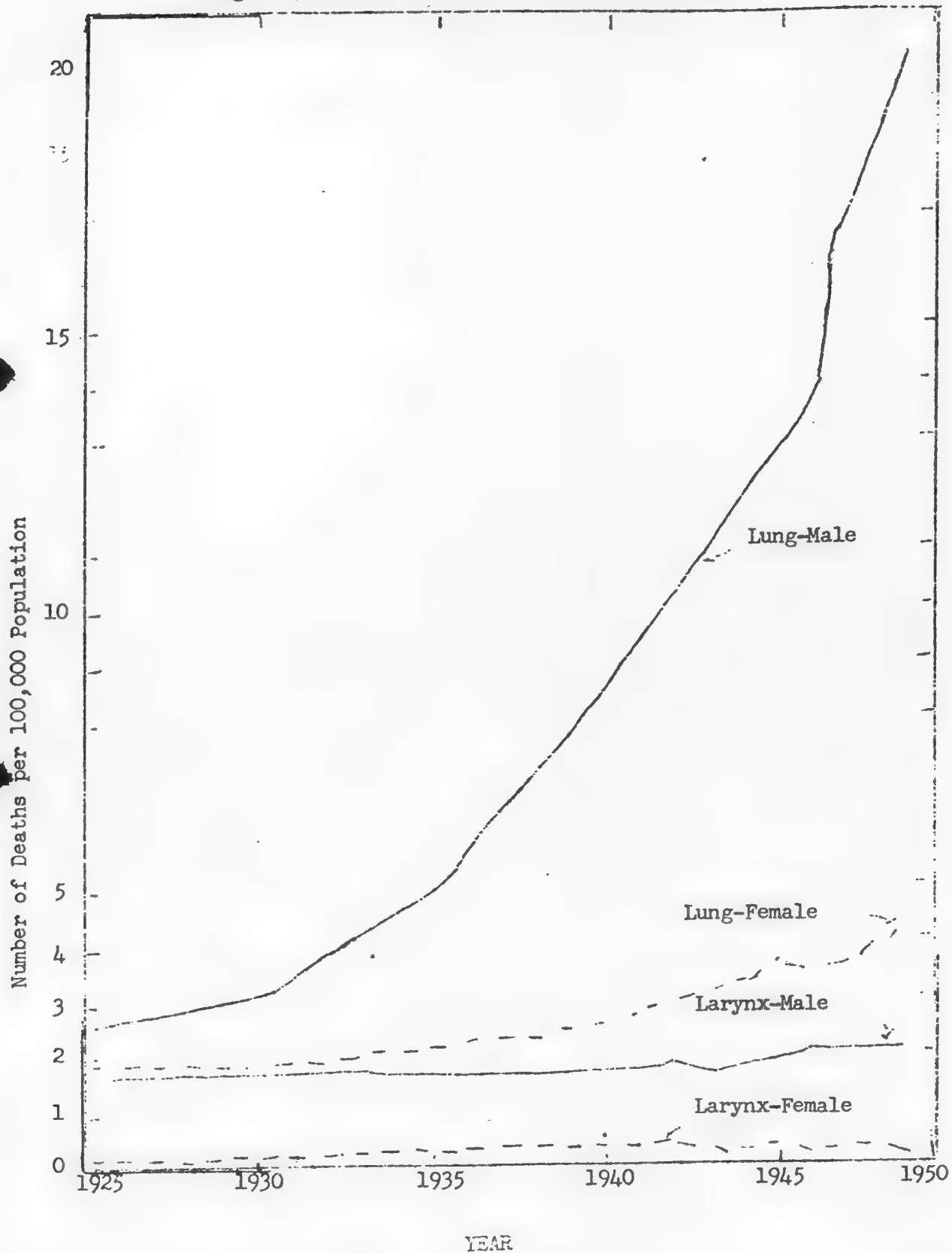
Schinz, Rosin and Sonti reported from Zurich, Switzerland, that the male mortality due to carcinoma of the lung between 1936 and 1944 was 9.8 per 100,000

Table 1.

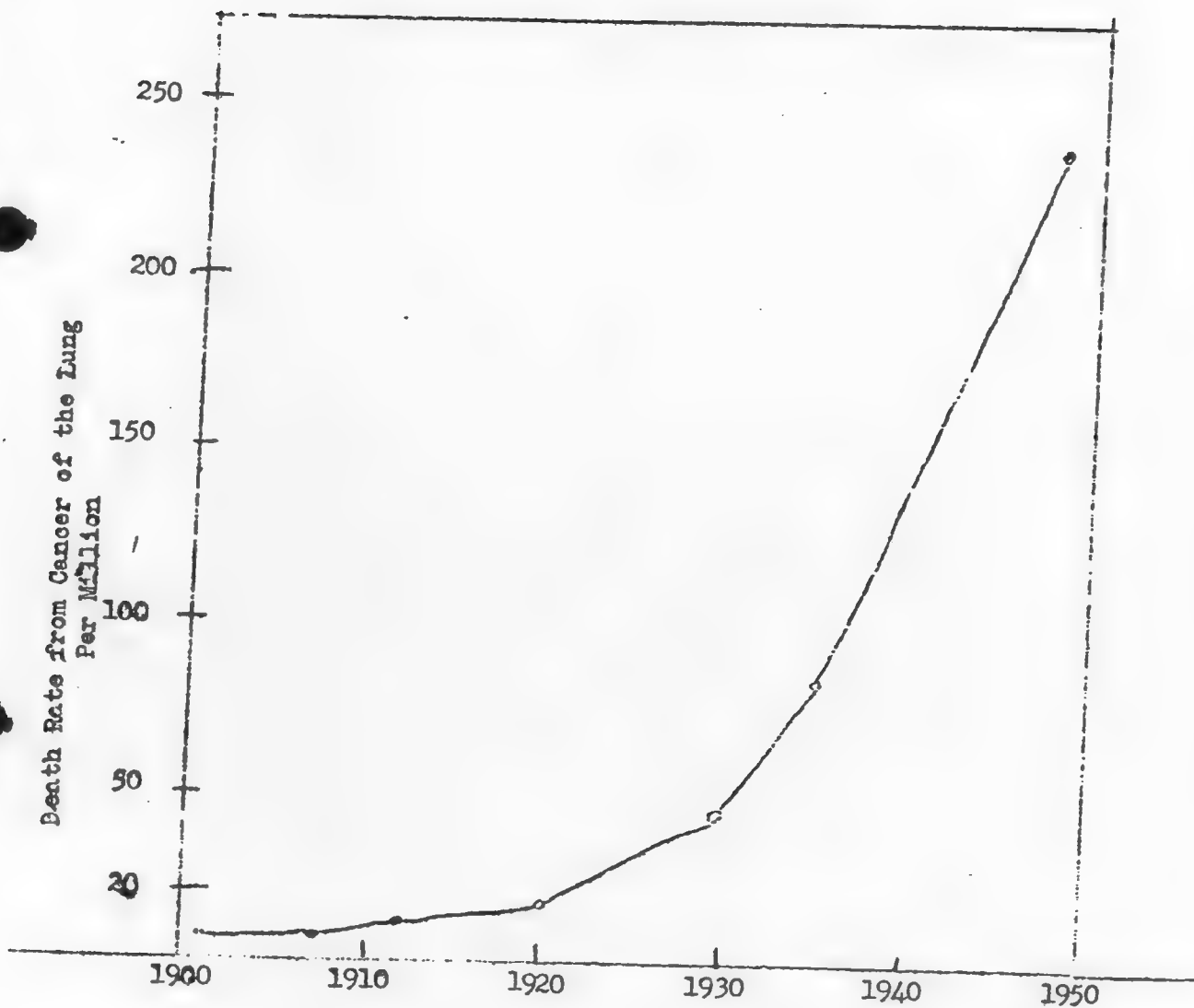
Annual Age Adjusted Increase of Frequency of Lung Cancer Mortality

| <u>Dorn</u> | <u>1914-1930</u> | <u>1931-1940</u> | <u>Potter</u> | <u>1933-1944</u> |
|-------------|------------------|------------------|---------------|------------------|
| Males | 10.5% | 8.5% | | 5.8% |
| Females | 8.0% | 2.5% | | 2.0% |

Trend in Mortality From Respiratory Cancer,
Lung compared with Larynx, by Sex, 1925-1928



Lung Cancer Death Rates
for England and Wales



The rates are based on three year averages for all years
except 1947

Doll, R. & Hill, A.B.: Brit. Med. J. 1: 739 (1950)

whereas it was 2.7 per 100,000 from 1896 to 1905. Brandt noted that in Riga, Latvia, the lung cancer incidence rose from 1.5 percent of all cancers found at autopsy in 1901 - 1905 to 10.5 percent in 1921 - 1925. Simross' analysis of the post-mortem material of Goettingen, Germany, yielded similar information, since cancer of the lung constituted 2.6 percent of all cancer in 1906 to 1912, while they were 9.8 percent in 1927 - 1931.

According to a report of von Glinski in 1939, 6.82 percent of all cancers seen at autopsy in Stettin, Germany, involved the lungs. Clemmesen evaluating official Danish mortality figures noted an apparent rise in lung cancer among males, whose attack rate increased from 5 per 100,000 in 1931 in Copenhagen to about 25 per 100,000 living in 1945, while the corresponding figures for females amounted to 4 and 7 per 100,000 respectively. The observations of Husted and Billman on Danish post-mortem material confirmed this trend. The investigations of Henschen on the autopsy material of Stockholm hospitals (Sweden) also revealed a progressive increase of lung cancer during the period 1900 to 1946. During the period 1900 - 1909, 1.6 percent of all cancers affected the lungs, during 1910 to 1919 the incidence figure stood at 2.1 percent and had risen to 5.85 percent for the period of 1920 to 1929. In the autopsy material of the St. Erik's Hospital in Stockholm, 9.9 percent of all cancers observed during 1937 to 1946 were located in the lung. While Casole reported in 1927 that among 2,658 necropsies performed in Padua during 1914 and 1925 there were two cancers of the lung (0.07 percent) and among 11,968 autopsies made during 1910 - 1925 in Milan, there were 15 pulmonary cancers (0.13 percent), Fabris noted in 1938, 150 lung cancers among 10,000 necropsies seen in ten years in Venice (1.5 percent).

The statistical studies of Stocks have demonstrated that also in England and Wales there has occurred a phenomenal increase in the frequency of lung cancer during the last several decades (fig. 2). Cancer of the lung in

England and Wales had standardized death rates of 1.1 for males and 0.7 for females during 1901 - 1920 and 10.6 for males and 2.5 for females in 1936 - 1939, which are almost identical with those recorded by Schinz for Zurich. These observations are in a general way confirmed for Great Britain by the studies of Kennaway; Kennaway and Kennaway; Heady and Kennaway; and Cheoseman as well as by those of Bonser. This investigator noted that there was a noticeable increase in the incidence of intrathoracic cancers at post-mortem at the Leeds General Infirmary during 1928 to 1937, following a long period (1891 - 1927) of relatively steady incidence, for which (1894 - 1928) also Passey and Holmes, using post-mortem material of various hospitals, did not observe any significant changes. Heady and Kennaway pointed out that there occurred a ninefold increase of cancer of the lung in men in England and Wales between 1928 and 1947, according to data of death certificates.

The evidence based on death rates from lung cancer and on autopsy observations was confirmed by the results of recent morbidity studies on cancer of the respiratory tract for eight metropolitan centers in the United States (Dorn; Warren; Grodowitz; Cutler; Marcus). The surveys conducted first in 1937 and repeated in 1947, showed for all metropolitan centers a consistent and often considerable increase in the morbidity rates of both cancers of the lung and, to a lesser extent, of the larynx (table 2). The rise in larynx cancer morbidity in the urban areas in the United States demonstrated by the surveys between 1937 and 1947 appears to be definitely more pronounced than the increase of crude larynx cancer death rates per 100,000 in the United States (1930: males, 1.42; females, 9.22; 1937: males, 1.70; females, 0.25) (Jackson and Jackson). These observations on the trend of death rates of larynx cancer in the United States are in relatively close agreement with those made in England and Wales (Kennaway and Kennaway) which stood in 1932 at 4.4 per 100,000 males and at 1.1 for 100,000 females, while they were in

Table 2.

Incidence of Respiratory Cancer, 1937 and 1947
Morbidity Rates for Eight Metropolitan Centers, by Sex
per 100,000 population*

| Primary Site and City | Males | | | Females | | | Total | | |
|--------------------------|-------|------|---------------------|---------|------|---------------------|-------|------|---------------------|
| | 1937 | 1947 | Percent Increase | 1937 | 1947 | Percent Increase | 1937 | 1947 | Percent Increase |
| <u>Bronchus and Lung</u> | | | | | | | | | |
| Atlanta | 5.0 | 13.4 | 168 | 1.0 | 5.0 | 400 | 2.9 | 8.9 | 207 |
| New Orleans | 13.1 | 39.1 | 198 | 2.8 | 4.2 | 50 | 7.6 | 20.8 | 174 |
| Dallas | 5.9 | 29.0 | 392 | 0.5 | 6.4 | 1180 | 3.1 | 17.2 | 455 |
| Birmingham | 4.5 | 18.9 | 320 | 2.1 | 3.9 | 86 | 3.3 | 11.0 | 233 |
| Denver | 9.1 | 21.9 | 141 | 4.2 | 8.1 | 93 | 6.6 | 14.8 | 124 |
| San Francisco | 15.6 | 34.3 | 120 | 3.9 | 8.1 | 108 | 9.8 | 20.8 | 112 |
| Chicago | 13.3 | 29.5 | 122 | 4.3 | 7.0 | 63 | 8.8 | 18.0 | 105 |
| Pittsburgh | 9.7 | 26.1 | 169 | 4.9 | 5.5 | 12 | 7.3 | 15.6 | 114 |
| Detroit | 12.6 | 32.0 | 154 | 2.3 | 5.7 | 148 | 7.5 | 19.0 | 159 |
| <u>Larynx</u> | | | | | | | | | |
| Atlanta | 1.4 | 4.0 | 186 | 0.3 | 0.3 | | 0.9 | 2.0 | 122 |
| New Orleans | 11.3 | 14.9 | 32 | 0.4 | 1.0 | 150 | 5.6 | 7.6 | 36 |
| Dallas | 3.2 | 5.3 | 66 | 1.5 | 0.4 | 73 | 2.3 | 2.7 | 17 |
| Birmingham | 1.4 | 4.0 | 186 | 0.0 | 1.3 | | 0.7 | 2.6 | 271 |
| Denver | 2.0 | 4.1 | 105 | 0.0 | 0.0 | | 0.9 | 2.0 | 122 |
| San Francisco | 4.5 | 8.8 | 96 | 0.2 | 0.8 | 300 | 2.4 | 4.6 | 92 |
| Chicago | 6.7 | 7.0 | 4 | 0.4 | 0.6 | 50 | 3.5 | 3.7 | 6 |
| Pittsburgh | 4.4 | 8.0 | 82 | 0.4 | 0.8 | 100 | 2.4 | 4.4 | 83 |
| Detroit | 3.5 | 6.4 | 83 | 0.4 | 0.3 | -25 | 2.0 | 3.4 | 70 |

*Biometrics Section
National Cancer Institute

1949, 4.1 for males and 1.3 for females.

It is apparent from these data that whatever factors may be responsible for the remarkable rise in the frequency of lung cancer, they have not - or to a much lesser degree - affected the incidence of cancer of the larynx.

If all due and liberal allowance is made for the progressive aging of the population since 1900, for an increased awareness of the medical profession of pulmonary cancer, for improved diagnostic facilities, for better recording systems, and for decreased deaths from other diseases than cancer, there still remains a considerable balance of lung cancers which cannot plausibly be accounted for by these factors. Surely, improvement in the medical proficiency of the pathologic diagnosis of lung cancer can scarcely be advanced as a factor of importance, since the histologic diagnosis of lung cancer is in general not a difficult task and has been practiced with efficiency and competence for many decades in the well-organized pathological institutes of Europe, which serviced the large public hospitals with their sociologically and economically relatively stable patient populations. The numerous objections voiced by Machlin on statistical grounds against the claim of an increase of lung cancers, pertain to only a minor degree to those institutes. Since fundamental changes in the biologic composition and constitution of the population groups involved did not take place within a few decades during which the increase in lung cancer frequency occurred, there remain only alterations in environmental factors related to modern industrialization and living conditions which plausibly might have provided the main causation of this development (Boycott).

2. Geographical Distribution.

The concept that exogenous factors, entering the human environment some 75 to 50 years ago and acting in increasing intensity, are responsible for the recent increase in lung cancers is supported by additional epidemiologic

observations, related to the geographical distribution of pulmonary cancers. The data on the incidence of respiratory cancer, 1937 and 1947, in eight metropolitan centers (table 2) reveal striking differences in the lung cancer morbidity rates of different centers, the extremes being 39.1 per 100,000 population, males, 1947, in New Orleans and 13.4 per 100,000 in Atlanta.

The percentages of increase in frequency for these communities also were far from uniform. Since the most marked discrepancies on these two points occur among metropolitan areas (New Orleans and Atlanta) located in the same part of the country (Southern States), it is most unlikely that differences in the genetic-biologic composition of the populations or fundamental variations in the smoking habit between these populations can be responsible for them. It is, therefore, much more probable that occupational, industrial or other environmental factors related to living conditions account for these regional variations.

Similar variations appear if cancer mortality data for different areas of States are analyzed. When Colorado, for instance, is divided into three regions according to predominating types of occupational activities (eastern part with agriculture, central part with industry, western part with mining and ranching) (fig. 3), it appears that the highest lung cancer death rate exists in the central, industrialized portion (50 per 100,000 male deaths) while the lowest is found in the agricultural area (30 per 100,000), with the western mining regions occupying an intermediary position (32). Seelig and Benignus also demonstrated that lung cancer death rates for 1930 to 1934 were higher in the urban areas than in the rural ones of the forty-eight States.

Similar state-wide discrepancies in the distribution of pulmonary cancer appear, if the lung cancer death rates of the forty-eight States are compared (table 3).

SPECIFIC CANCER DEATHS IN MALES

MINING
RANCHING

INDUSTRY

AGRICULTURE

DEATH RATE
ALL CANCERS-147
PER 100,000
SKIN-15
RESPIRATORY-32

DEATH RATE
ALL CANCERS-200
PER 100,000
SKIN-15
RESPIRATORY-50

DEATH RATE
ALL CANCERS-150
PER 100,000
SKIN-13
RESPIRATORY-30

COLORADO - 1943 - 1949

Table 3.

Lung Cancer Death Rates in the 25 States of the United States
in 1946 and 1948
Crude Death
Rates per 100,000

| Industrialized States | | |
|-----------------------|------|------|
| State | 1946 | 1948 |
| Connecticut | 8.5 | 11.1 |
| Illinois | 8.1 | 8.2 |
| Maryland | 6.5 | 8.4 |
| Massachusetts | 10.4 | 10.2 |
| Michigan | 5.7 | 7.1 |
| New Hampshire | 7.4 | 10.1 |
| New Jersey | 9.7 | 9.7 |
| New York | 10.2 | 11.9 |
| Ohio | 6.0 | 7.9 |
| Pennsylvania | 6.7 | 8.4 |
| Rhode Island | 8.7 | 7.4 |

| States with Regional Industrialization | | |
|---|------|------|
| State | 1946 | 1948 |
| Florida | 6.8 | 7.4 |
| Louisiana | 6.6 | 8.5 |
| Missouri | 7.3 | 9.4 |
| Montana | 10.0 | 8.8 |
| Nebraska | 5.7 | 8.0 |

| Agricultural States | | |
|---------------------|------|------|
| State | 1946 | 1948 |
| Alabama | 4.0 | 5.1 |
| Arkansas | 3.6 | 5.4 |
| New Mexico | 2.6 | 3.0 |
| North Carolina | 3.1 | 4.0 |
| North Dakota | 5.6 | 4.1 |
| Oregon | 4.1 | 4.4 |
| South Carolina | 3.6 | 3.7 |
| Washington | 5.1 | 4.2 |
| Wyoming | 4.9 | 3.9 |

The death rates for the year 1946 were taken from "The American Cancer Society, Inc., 1949, Cancer Death Rates for each State in the United States by Site"; those for the year 1948 were produced by the National Office of Vital Statistics (Rigdon and Kirchoff).

BIBLIOGRAPHY

Occupational and Environmental Pulmonary Cancer

With Special Reference to Pneumoconiosis

W. C. Hueper

- Abraham, H., *Asphalts and Allied Substances*. 5th Edition, Vol.2, d. Van Nordstrand Co., New York, 1945, p. 481.
- Adler, J., *Growths of the Lungs and Bronchi*. Longmans, Green and Co., New York, 1912.
- Agricola, G., *De re metallica*, Basel, 1597.
- Akatsuka, K. and Fairhall, L. T., *J. Ind. Hyg.* 16: 1, 1934.
- Aldridge, W. N., Barnes, J. M. and Denz, F. A., *Brit. J. of Exper. Path.*, London, 30: 375, 1949.
- Aldridge, W. N., Barnes, J. M. and Denz, F. A., *Brit. J. of Exper. Path.*, 31: 473, 1950.
- Allen, M. L., *J. Ind. Hyg.* 16: 346, 1934.
- Alwens, W., *Muench. med. Wchnschr.* 82: 1727, 1935.
- Alwens, W., *Proc. 3th Internat. Kongr. f. Unfallmed. u. Berufsk.*, Budapest, 1938. Published, Georg Thieme, Leipzig, 1939, p. 973.
- Alwens, W. and Jonas, W., *Arch. f. Gewerbepath. u. Gewerbehyg.* 7: 3, 1936.
- Alwens, W. and Jonas, W., *Arch. f. Gewerbepath. u. Gewerbehyg.* 7: 532, 1936-1937.
- Alwens, W. and Jonas, W., *Acta Unio internat. contra cancerum* 3: 103, 1938.
- Alwens, W., Bauke, E. E. and Jonas, W., *Arch. f. Gewerbepath. u. Gewerbehyg.* 7: 69, 1936.
- Alwens, W., Bauke, E. E. and Jonas, W., *Muench. med. Wchnschr.* 83: 485, 1936.
- American Cancer Society, Inc., 1949, *Cancer Death Rates for Each State in the United States by Site*.
- Amor, A. J., *J. Ind. Hyg.* 14: 216, 1932.
- Amor, A. J., *Occupation and Health*, International Labour Off., Geneva, Suppl. 1938.
- Amor, A. J., *3th Internat. Kongr. f. Unfallmed. u. Berufsk.* 2: 941, 1938.
- Anderson, C. S. and Dible, J. H., *J. Hyg.* 38: 135, 1938.

- Andrievskaya, Z. M. and Mislavskaya, M. M., *Gigiena I. Sanitariya*.
Moscow, No. 5: 28, 1949.
- Anissimova, V., *Am. J. Cancer* 36: 229, 1939.
- Ann. Rep. Chief Insp. of Factories, 1944. H. M. Stationary Office,
London, p. 55.
- Ann. Rep. Chief Insp. of Factories, 1947. H. M. Stationary Office,
London, Medical Section, pp. 6, 12, 76, 79.
- Ann. Rep. Chief Insp. of Factories, 1948. H. M. Stationary Office,
London, pp. 90, 91, 93.
- Araki, M. and Mure, K., *Gann* 40: 76, 1949.
- Arguello, R. A., Tollo, E. E., Macola, B. A. and Manzano, L., *Rev. Fac.
cienc. med. Univ., Cordoba* 8: 409, 1950.
- Arhelger, S. W. and Kremen, A. J., *Surg.* 30: 977, 1951.
- Arkin, A. and Wagner, D. H., *J.A.M.A.* 106: 587, 1936.
- Armit, H. W., *J. Hyg.* 7: 525, 1907.
- Armit, H. W., *J. Hyg.* 8: 565, 1908.
- Arnstein, A., *Wien. klin. Wchnschr.* 26: 748, 1913.
- Arnstein, A., *Verhandl. d. deutsch. path. Gesellsch.* 16: 332, 1913.
- Arrigoni, A., *Med. d. Lavoro* 24: 461, 1933.
- Aske-Upmark, E., *Acta Path. et microbiol. Scandinav.* 9: 159, 1932.
- Attinger, E., *Oncologia, Basel* 3: 129, 1950.
- Aub, J. C. and Grier, R. S., *J. of Ind. Hyg. & Toxicol.* 31: 123, 1949.
- Aucke, Der Lungenkrebs in den Schneeberger. *Ein fall- von Lymphosarcoma
fibromatode. Inaug. Diss. München, 1884.*
- Auerbach, C., *Science* 110: 419, 1949.
- Auld, S. J. M., *J. Inst. of Petroleum* 36: 235, 1950.
- Baader, E. W., *Arch. f. Gewerbepath. u. Gewerbehyg.* 4: 101, 1932-1933.
- Baader, E. W., quoted by Bauer, K. H., *Verhandl. d. deutsch. path.
Gesellsch.* 30: 239, 1937.
- Baader, E. W., in Adam, C. and Auler, H., *Monograph, Neuere Erg. a. d.
Geb. d. Krebskrankheiten, S. Hirzel in Leipzig, 1937, pp. 104-128.*

- Baader, E. W., Arch. Mal. Prof. 1: 104, 1938.
- Baader, E. W., Klin. Wchnschr. 18: 522, 1939.
- Baader, E. W., Personal communication, 1951.
- Baader, E. W., Verhandlungen der Deutschen Gesellschaft für innere Medizin 57. Kongr., 322, 1951.
- Baetjer, A. M., Arch. of Ind. Hyg. & Occup. Med. 2: 487, 505, 1950.
- Bagg, H. J., Am. J. Cancer 26: 69, 1936.
- Bagg, H. J., Science, Suppl. 85: 92, 1937.
- Balestra, G. and Molfino, F., Rassegna di med. indust. 13: 5, 1942.
- Barnard, C. I., The Rockefeller Foundation - A Review for 1949.
- Barnes, J. M., Proc. 9th Internat. Cong. on Ind. Med., London, p. 630, 1948.
- Barnes, J. M., Lancet 1: 463, 1950.
- Barnes, J. M., Denz, F. A. and Sissons, H. A., Brit. J. Cancer 4: 212, 1950.
- Barrie, H. J. and Harding, H. E., Brit. J. of Ind. Med. 4: 225, 1947.
- Barsky, M. H., New York State J. of Med. 37: 1031, 1937.
- Barták, F. and Tomečka, M., Proc. 9th Internat. Cong. on Ind. Med., London, p. 744, 1948.
- Bauer, K. H., Arch. f. klin. Chir. 189: 123, 1937.
- Bauer, K. H. Erkrankungen von Braunsteinarbeiters an Lungenerstzuendung, Thesis. Univ. Berlin, K. and R. Hoffmann, Berlin, 1938, 48 pages.
- Bauer, R., Strahlentherapie 64: 249, 1939.
- Bauer, K. H., Das Krebsproblem, Publisher Springer-Verlag, Berlin, 1949, 758 pages.
- Bauer, J. T. and Schraer, P. H., Am. J. Path. 16: 657, 1940.
- Bauke, E. and Alwens, W., Verhandl. d. deutsch. Gesellsch. f. inn. Med. 48: 199, 1936.
- Bauke, E. and Jonas, W., Muench. med. Wchnschr. 12: 485, 1936.
- Bayer, O., Arch. f. Gewerbepath. u. Gewerbehyg. 9: 592, 1938-1939.
- Beeler, T. T., Jr. and Ireys, N. S., Dis. of Chest 18: 61, 1950.

- Beerman, Herman, Am. J. of Med. Sci. 221: 462, 1951.
- Behounek, F., Physik. Ztschr. 28: 333, 1927.
- Behounek, F., 4th Internat. Radiologen Kongr., Zurich 2: 413, 1934.
- Behounok, F. and Fort, M., Strahlentherapie. 70: 487, 1941.
- Beintker, E., Med. Welt. 9: 364, 1935.
- Belt, T. H., Frankfurt. Ztschr. f. Path. 42: 170, 1931-1932.
- Berblinger, W., Klin. Wchnschr. 4: 913, 1925.
- Berblinger, W., Med. Klin. 27: 1337, 1931.
- Berenblum, I., Arch. of Path. 38: 233, 1944.
- Berenblum, I. and Schoental, R., Brit. J. of Exper. Path. 24: 232, 1943.
- Berenblum, I. and Schoental, R., Brit. J. of Cancer 1: 157, 1947.
- Berg, R., Jr. and Burford, T. H., J. Thoracic Surg. 20: 418, 1950.
- Bergmann, M., Shatz, B. A. and Flance, I. J., J.A.M.A. 138: 798, 1948.
- Bergmann, M. and Graham, E. A., J. Thoracic Surg. 22: 549, 1951.
- Berlinger, W., Med. Klin. 27: 1337, 1931.
- Berman, I. L. and Ernest, E. P., Ind. Med. & Surg. 19: 229, 1950.
- Berry, J. W., Am. Rev. Tuberc. 57: 557, 1948.
- Bertrand, G. and Vladesco, R., Compt. rend. Acad. d. sc. 173: 176, 1921.
- Bertrand, G. and Serbescue, P., Ann. Inst. Pasteur. 53: 10, 1934.
- Best, G. E., Arch. Hyg. & Occup. Med. 5: 517, 1952.
- Best, G. E., Am. Ind. Hyg. Assn. Quarterly 13: 62, 1952.
- Betke, H., Jahrb. d. preuss. Gewerbeärzte, p. 84, 1933.
- Beutel, A. and Woldrich, A., Ztschr. f. Krebsforsch. 34: 109, 1931.
- Beyreuther, H., Virchows Arch. f. path. anat. 250: 230, 1924.
- Bhattacharya, R., J. Soc. Chem. Ind. 54: 82, 1935.
- Bidstrup, P. L., Archives Belges De Médecine Sociale, Hygiene, Médecine Du Travail et Médecine Légale, No. 8: 500, 1950.

- Bidstrup, P. L., Brit. J. Ind. Med. 8: 302, 1951.
- Birkholz, Arch. f. Ohren-, Nasen- und Kehlkopfheilk 111: 123, 1924.
- Black, C. E., Arch. of Path. 35: 366, 1943.
- Blina, L. V., La Riforma Medica, Naples 44: 1516, 1928.
- Bloomfield, J. J. and Blum, W., Public Health Rep., U.S.P.H.S. 43:2330, 1928.
- Bloomfield, J. J., Trasko, V. M., Sayers, R. R., Page, R. T. and Payton, M.F.,
Public Health Bull. No. 259, 1940.
- Blumenthal, F., Ztschr. f. Krebsforsch. 30: 314, 1929-1930.
- Bodmer, H. Kallos, P., Arch. f. Ohren-, Nasen- und Kehlkopfheilk 136: 40,
1933.
- Boemke, F., Ber. 8th Internat. Kongr. f. Unfallmed. u. Berufsk., Frankfurt
a. M. 2: 1038, Georg Thieme, Leipzig, 1938.
- Bogen, E. and Loomis, R. N., Am. J. Cancer 16: 1515, 1932.
- Bohme, A., Klin. Wchnschr. 15: 731, 1936.
- Bohme, A., Arch. f. Gewerbepath. u. Gewerbehyg. 11: 433, 1942.
- Bohne, cited by Wedler.
- Bohnenkamp, H., 8th Internat. Kongr. f. Unfallmed. u. Berufsk., 1938,
p. 1069.
- Bohrod, M. G., Arch. Path. 10: 179, 1930.
- Bolen, H. L., J. Ind. Hyg. & Toxicol. 25: 215, 1943.
- Bonser, G. M., J. Hyg. 28: 340, 1929.
- Bonser, G. M., J. Hyg. 34: 218, 1934.
- Bordas, F. B., The Prevention of Cancer. Report, the Council Supérieur
d'Hygiène publique de France, 1929.
- Borst, M., J. F. Bergmann, Wiesbaden Vol. I, p. 80: Vol. II, p. 754, 1902.
- Bourne, H. G. and Yee, H. T., Ind. Med. & Surg. 19: 563, 1950.
- Bourne, H. G., Jr. and Rushin, W. R., Ind. Med. & Surg. 19: 568, 1950.
- Bourret, J. and Fraisse, H., J. de med. de Lyon. 29: 123, 1948.
- Boycott, A. E., Lancet 2: 959, 1932.

- Boyd, M. A., *Lancet* 2: 60, 1887.
- Brandeis, W. W., *J.A.M.A.* 102: 1204, 1934.
- Brandt, M., *Deutsche med. Wchnschr.* 53: 1824, 1927.
- Brandt, A., *Ztschr. f. Krebsforsch.* 47: 108, 1938.
- Breslin, A. J., *Am. Ceram. Soc. Bull.* 30: 395, 1951.
- Breslow, L., *Environmental Aspects of Pulmonary Cancer. Chronic Disease Service, Calif. Dept. of Public Health*, 1950.
- Breslow, L., *California's Health, St. Dept. of Public Health* 9: 1, 1951.
- Brezina, E., *Internationale Übersicht über Gewerbekrankheiten. Publisher, Julius J. Springer*, 1929.
- Brezina, E., *Schrift. a. d. Gesamtgeb. d. Gewerbehyg., Springer, Berlin*, 1931, No. 36: reviewed in *J. Ind. Hyg.* 14: 237, 1932.
- Bridge, J. C., *Ann. Rep. Gen. Med., Inspector of Factories*, p. 74, 1931.
- Bridge, J. C., 3rd Chapt. *Ann. Rep. of the Chief Insp. of Factories*, 1935. H. M. Stationery Office, London, 1936.
- Bridge, J. C., *Ann. Rep. of the Chief Insp. of Factories and Workshops*, 1936. H. M. Stationery Office, London, 1936.
- Bridge, J. C., *Ann. Rep. of the Chief Insp. of Factories*, 1939. H. M. Stationery Office 1: 57, 1941.
- Brinkmann, *Über Lungenkarzinom, Inaug. Diss., Leipzig*, 1924.
- Brockbank, W., *Quart. J. Med.* 1: 31, 1932.
- Brockbank, E. M. and Stopford, J. S. B., *Brit. Med. J.* 2: 993, 1927.
- Brown, E. V. and Van Winkle, W., Jr., *J.A.M.A.* 140: 1024, 1949.
- Bruce, R. A., Lovejoy, F. W., Jr., Brothers, G. B. and Velasquez, T., *U. S. Atomic Energy Comm. AECD-2391*, 1948.
- Brunner, A., *Schweiz. med. Wchnschr.* 81: 653, 1951.
- Bryan, F. A. and Silverman, I. B., *U. S. Navy Med. News Letter* 14: 22, 1949.
- Buchan, R. F., *Occup. Med.* 3: 439, 1947.
- Buckell, M., Garraud, J., Jupe, M. H., McLaughlin, A. I. G. and Perry, K., *Brit. J. Ind. Med.* 3: 78, 1946.

- Buckell, M. and Harvey, D. G., Brit. J. of Ind. Med. 8: 298, 1951.
- Buess, H., Helv. med. Acta 17: 104, 1950.
- Burckhardt, W., Arch. f. Dermat. u. Syph. 173: 262, 1936.
- Buttner, H. E., Ergebn. d. inn. Med. u. Kinderh. 58: 1, 1940.
- Buttner, H. E., and Lenz, E., Arch. f. Gewerbepath. u. Gewerbehyg. 7: 672, 1937.
- Butzengeiger, K. H., Klin. Wchnschr. 19: 523, 1940.
- Butzengeiger, K. H., Ärztl. Wchnschr. 4: 365, 1949.
- Byers, H. G., U. S. Dept. of Agric. Tech. Bull. No. 482, 1935; No. 530, 1936.
- Cadotsch, H., Arch. f. Ohren-, Nasen- und Kehlkopfheilk 157: 68, 1950.
- Cameron, G. R., J. Path. & Bact., London 59: 539, 1947.
- Caminta, B. H., Baum, W. S., Neal, P. A. and Schneiter, R. Public Health Bull. No. 297, U.S.P.H.S., 1947.
- Campbell, J. A., J. Path. & Bact. 36: 243, 1933.
- Campbell, J. A., Lancet 1: 233, 1934.
- Campbell, J. A., Brit. J. Exper. Path. 15: 287, 1934.
- Campbell, J. A., Brit. J. Exper. Path. 17: 146, 1936.
- Campbell, J. A., Brit. J. Exper. Path. 18: 215, 1937.
- Campbell, J. A., J. Ind. Hyg. & Toxicol. 19: 449, 1937.
- Campbell, J. A., Brit. J. Exper. Path. 20: 122, 1939.
- Campbell, J. A., Brit. Med. J. 2: 275, 1940.
- Campbell, J. A., Brit. J. Exper. Path. 23: 191, 1942.
- Campbell, J. A., Brit. Med. J. 1: 217, 1942.
- Campbell, J. A., Brit. Med. J. 1: 179, 1943.
- Campbell, J. A., Brit. J. Exper. Path. 25: 46, 1944.
- Cannon, A. B., New York St. J. of Med. 36: 1, 1936.
- Cannon, P. R., J.A.M.A. 115: 2176, 1940.
- Cannon, P. R. and Walsh, T. E., J.A.M.A. 114: 251, 1940.

- Carozzi, L., Report Internat. Cancer Conf., London, 1928, p. 311.
- Carozzi, L., Occupation and Health, Vol.2, Internat. Labour Office, Geneva, 1930, p. 1058.
- Carozzi, L., Arch. d'electric. med. 42: 85, 118 and 155, 1934.
- Carozzi, L., Acta Unio internat. contra cancerum 2: 3, 1937.
- Carter, W. W., M. J. & Rec. 130: 125, 1929.
- Cartier, P., Arch. Ind. Hyg. & Occup. Med. 5: 262, 1952.
- Casole, G., L'Ospedale Maggiore, Milan 15: 227, 1927.
- Cass, J. W., Jr., Arch. Ind. Hyg. & Occup. Med., Chicago 3: 569, 1951.
- Castleden, L. I. M. and Hamilton-Paterson, J. L., Brit. Med. J. 2: 478, 1942.
- Cawston, F. G., Acta med. Scandinav. 99: 92, 1939.
- Chamberlain, C. W., Ind. Med. 3: 286, 1934.
- Charr, R., Am. J. Med. Sci. 194: 535, 1937.
- Cheeseman, E. A., Ulster Med. J. 19: 158, 1950.
- Chikamatsu, T., cited by Sugiura.
- Chikamatsu, T., Tr. Soc. path. Jap. 21: 244, 1931.
- Clark, E. and Graef, I., Am. J. Path. 11: 693, 1935.
- Clemmesen, J., Cancer and Occupation in Denmark, 1935-1939, Nordisk Forlag-Arnold Busck, Copenhagen, p. 75, 1941.
- Clemmesen, J. and Busk, T., Brit. J. Cancer 1: 253, 1947.
- Cohen, A. C., Dis. of Chest 15: 607, 1949.
- Cohen, J. B. and Ruston, A. G., Smoke - A Study of Town Air. Edward Arnold & Co., London, 1925.
- Conheim, cited by Dohnert, H. R., Ztschr. f. Krebsforsch. 47: 209, 1938.
- Cook, J. W., Hewett, C. I. and Hieger, I., J. Chem. Soc. 1: 395, 1933.
- Cooper, E. H., Med. Press 187: 397, 1933.
- Cooper, E. A., Bott., H. G., Cheesworth, H. D. and Tipson, R. S., Parts I & II.; Lamb, F. W. M. and Sanders, E. Part III.; Hirst, E. L. Part IV, J. of Hyg. 32: 293, 1932.

- Corcoran, W. J., Radiology 50: 780, 1948.
- Cormia, E. and Stewart, G., Canad. Med. Assn. J., Montreal 32: 270, 1935.
- Cramer, A., Bull. et mem. Soc. d. chirurgiens de Paris 46: 926, 1922.
- Cristol, Compt. rend. Acad. d. sc. 174: 887, 1922.
- Crombie, D. W., Blaisdell, J. L. and MacPherson, G., Canad. Med. Assn. J. 50: 318, 1944.
- Cruickshank, W. H., Memorandum, 1941.
- Cruickshank, C. N. D. and Squire, J. R., Brit. J. Ind. Med. 7: 1, 1950.
- Cureton, R. J. R., Brit. J. of Cancer 2: 249, 1948.
- Curtis, G. H., Arch. Dermat. & Syph., Chicago 64: 470, 1951.
- Cutler, S. J., Cancer Morbidity Series 1, U.S.P.H.S., N.C.I., 1950, 43 pages.
- Cutler, S. J., Cancer Morbidity Series 5, U.S.P.H.S., N.C.I., 1951, 46 pages.
- Cutter, H. C., Faller, W.W., Stocklen, J. B. and Wilson, W. L., J. Ind. Hyg. & Toxicol. 31: 319, 1949.
- Czechoslovakian Commission, Official Report from Muench. med. Wehnschr. 82: 1666, 1935.
- Daff, M. E. and Kennaway, E. L., Brit. J. of Cancer 4: 173, 1950.
- Daff, M. E., Doll, R. and Kennaway, E. L., Brit. J. Cancer 5: 1, 1951.
- Dahlmann, J., Fortschr. a. d. Geb. d Rontgenstrahlen. 75: 628, 1951.
- Dankman, H. S., Arch. Ind. Hyg. & Occup, Med. 5: 228, 1952.
- Dassanayake, W. L. P., Brit. J. of Ind. Med. 5: 141, 1948.
- Davies, T. A. L., Brit. J. of Ind. Med., London 3: 111, 1946.
- Davis, C. and Grimes, O. F., Calif. Med. 74: 203, 1951.
- Davis, H. W., in Keiser, H. J. Minerals Yearbook 1945, Washington, D. C., 1947, p. 620.
- Davis, M. L., Ohio Public Works, p. 11 (Sept.) 1949.
- Dawson, K. B., Brit. J. Cancer 6: 22, 1952.
- De Balsac, F. H. and Feil, A., 8th Internat. Kongr. f. Unfallmed. u. Berufsk. 2: 985, 1938.
- DeLaet, cited by Maisin, J., Bruxelles med. 14: 544, 1934.

- DeNardi, J. M., Van Ordstrand, H. S. and Carmody, M. G., Ohio St. Med. J.
45: 567, 1949.
- Denker, A., in Denker and Kahler, Handbuch der Hals-Nassen-, und
Ohrenheilhunde, J. Springer, Berlin, Vol.5: p. 209, 1929.
- Deribere, M., Paris Med. 49: 591, 1950.
- Derischanoff, L., Ztschr. f. Krebsforsch. 36: 82, 1932.
- Derwitz, A. P., Arch. f. Dermat. 161: 1, 1930.
- Desmeules, R., Rousseau, L., Giroux, M. and Sirois, A., Laval Medical
6: 97, 1941.
- Desmeules, R., Rousseau, L., Giroux, M. and Sirois, A., Sem. Hop.
23: 1820, 1947.
- Dible, J. H., Lancet 2: 982, 1934.
- Dissmann, E., Ztschr. f. Krebsforsch. 36: 563, 1932.
- Division of Industrial Hygiene, Cadmium Poisoning, Public Health Report
57: 601, 1942.
- Dixon, F. W., J.A.M.A. 93: 837, 1929.
- Doenecke, F., Frankfurt. Ztschr. f. Path. 42: 161, 1931-1932.
- Doese, M., Arch. f. Gewerbepath. u. Gewerbehyg. 8: 501, 1938.
- Döhnert, H. R., Ztschr. f. Krebsforsch. 47: 209, 1938.
- Doig, A. T. and McLaughlin, A. I. G., Lancet 1: 789, 1948.
- Doll, R. and Hill A. B., Brit. Med. J. 2: 739, 1950.
- Domenici, cited by Koelsch, Arch. f. Gewerbepath. u. Gewerbehyg.
5: 454, 1935.
- D'Onofrio, V. and Passeri, A., Rassegna di med. indust., Turin 19:
276, 1950.
- Dorn, H. F., Public Health Rep. 58: 1265, 1943.
- Dorn, H. F., Public Health Rep. 59: 33, 65, 97, 1944.
- Doubrow, S., Paris med. 1: 287, 1931.
- Downing, J. G., J.A.M.A. 125: 196, 1944.
- Downing, J. G., J.A.M.A. 148: 245, 1952.

- Dreessen, W. C., Dalla Valle, J. M., et al. Public Health Bull. No. 241: 68, 1938.
- Dreessen, W. C., Brinton, H. P., Keenan, R. G., Thomas, T. R. and Place, E. H., Public Health Rep. 63: 488, 1948.
- Dreyfus, T. R., Ztschr. f. klin. Med. 130: 256, 1936.
- Drinker, K. R., Fairhall, L. T., Ray, G. B. and Drinker, C. K., J. of Ind. Hyg. 6: 307, 1924.
- Drinker, C. K. and Fairhall, L. T., Internat. J. Med. & Surg. 46: 553, 1933.
- Druckrey, H. and Schmähl, D., Ztschr. f. Naturforschg., in press. (1952).
- Drymalski and Sweany, Am. Rev. Tuberc. 58: 203, 1948.
- Dublin, L. I. and Vane, R. J., Metropolitan Life Insurance Co. Survey 1937-1939.
- DuBray, E. S., J.A.M.A. 108: 383, 1937.
- Dudley, H. C., Public Health Rep. 53: 281, 1938.
- Dudley, H. C., and Miller, J. W., J. Ind. Hyg. & Toxicol. 23: 470, 1941.
- Duguid, J. B., Lancet 2: 111, 1927.
- Dundon, C. C. and Hughes, J. P., Am. J. of Roentgenol. and Radium Therapy 63: 797, 1950.
- Dungal, N., Lancet 2: 245, 1950.
- Dunner, L., Brit. J. Radiol. 21: 182, 1948.
- Dunner, L. and Bagnall, J. T., Brit. J. Radiol. 19: 165, 1946.
- Dunner, L. and Bagnall, D. J. T., Brit. J. Radiol. 22: 573, 1949.
- Dutra, F. R., Am. J. of Path. 24: 1137, 1948.
- Dutra, F. R., Arch. Dermat. & Syph., Chicago 60: 1140, 1949.
- Dutra, F. R., Arch. of Ind. Hyg. & Occup. Med. 3: 81, 1951.
- Dutra, F. R., Largent, E. J. and Roth, J. L., Atomic Energy Comm., Unclassified Document 1272, 13 pages.
- Dutra, F. R., Largent, E. J. and Roth, J. L., Arch. of Path. 51: 473, 1951.
- Dutra, F. R., Largent, E. J., Cholak, J., Hubbard, D. M. and Roth, J. L., Arch. of Ind. Hyg. & Occup. Med. 4: 65, 1951.

- Earle, J., The Chirurgical Works of Percival Pott, 3 Vols., 2nd Ed., 3, p. 182, 1808.
- Editorial, J.A.M.A. 87: 1397, 1926.
- Editorial, Lancet 2: 1478, 1936.
- Editorial, Lancet 2: 27, 1951.
- Editorial, Lancet 2: 30, 1952.
- Edmundson, F., J. Invest. Dermat. 17: 17, 1951.
- Ehrismann, O., Ztschr. f. Hyg. u. Infektionskr. 117: 662, 1935.
- Ehrismann, O., Ztschr. f. Hyg. u. Infektionskr. 122: 166, 1939.
- Eisenbud, M., Berghout, C. F. and Steadman, L. T., J. Ind. Hyg. & Toxicol. 30: 281, 1948.
- Eisenbud, M., Wanta, R. C., Dustan, C., Steadman, L. T., Harris, W. B. and Wolf, B. S., J. Ind. Hyg. & Toxicol. 31: 282, 1949.
- Elkins, H. B., Chemistry of Industrial Toxicology. Publisher, John Wiley & Sons, Inc., New York, 1950, 406 pages: Chapman & Hall, Limited, London.
- Elmore, D. T., Gulland, F. M., Jordan, D. O. and Taylor, H. F. W., Biochem. J. 42: 308, 1948.
- Elstad, D., Nord. Med. 3: 2527, 1939.
- Engelhardt, W. E. and Mayer, R. L., Arch. f. Gewerbepath. u. Gewerbehyg. 2: 140, 1931.
- Engelstad, R. B., Acta radiol., Suppl. 19, 1934.
- Enger, Inaug. Diss., Leipzig, 1923.
- Environment and Health, Fed. Sec. Agency, U.S.P.H.S., 152 pages, 1951.
- Erfan, N., Transactions Royal Soc. Trop. Med. and Hyg., London 42: 109, 1948.
- Evans, E. H., Lancet 2: 368, 1945.
- Evans, R. D. and Goodman, C., J. Ind. Hyg. & Toxicol. 22: 89, 1940.
- Ewing, J., Neoplastic Diseases, Publisher, W. B. Sanders Co., Philadelphia, Pa., 3rd Edition, 1928.
- Fabris, A., Acta Unio internat. contra cancerum 3: 130, 1938.

- Fairhall, L. T., Industrial Toxicology. Publisher, The Williams & Wilkins Co., Baltimore, Md., 1949, 483 pages.
- Fairhall, L. T. and Neal, P. A., Nat. Inst. of Health Bull. No. 182, U.S.P.H.S., 1943.
- Fairhall, L. T., Castberg, H. T., Carrozzo, N. J. and Brinton, H. P., Occup. Med. 4: 371, 1947.
- Fairhall, L. T. and Hyslop, F., Public Health Rep., Suppl. No. 195, 1947.
- Fairhall, L. T., Keenan, R. G. and Brinton, H. P., Public Health Rep. 64: 485, 1949.
- Falin, L. I. and Gromzewa, K. E., Am. J. Cancer 36: 233, 1939.
- Falin, L. I. and Gromzewa, K. E., Virchows Arch. f. path. Anat. 306: 300, 1940.
- Falin, L. I. and Gromtseva, K. E., Arkhiv Biologicheskikh Nauk, Leningrad 60: 86, 1940.
- Falin, L. I. and Anissimowa, W. W., Ztschr. f. Krebsforsch. 50: 339, 1940.
- Falk, H. L., Steiner, P. E., Goldfein, S., Breslow, A. and Hykes, R., Cancer Res. 11: 318, 1951.
- Falk, H. and Steiner, P. E., Cancer Res. 12: 30, 40, 1952.
- Fed. Sec. Ag., Regional and Field Letter O.F.S., Washington, D.C., No. 833, Item A9, 1951.
- Feil, A., Semaine D. Hop. De Paris 6: 364, 1930.
- Feil, A., Presse méd. 43: 212, 1935.
- Feil, A., Rev. med. franc. 17: 745, 1936.
- Feil, M. A., Presse méd. 47: 1133, 1939.
- Fenn, G. K., Arch. Ind. Hyg. & Occup. Med. 3: 571, 1951.
- Ferenczy, K. and Matolcsy, T., Wien. klin. Wchnschr. 40: 618, 1927.
- Ferrari, E., Muench. med. Wchnschr. 80: 942, 1933.
- Filipo, D., Rassegna di med. indust. 5: 128, 1934.
- Fine, M. J. and Jaso, J. V., J.A.M.A. 104: 40, 1935.
- Fischer, A. W. and Fenster, E., Monatschr. f. Unfallh. 45: 158, 1938.

Fischer, H. G. M., Priestley, W., Eby, L. T., Wanless, G. G. and Rehner, J., Jr., Arch. Ind. Hyg. & Occup. Med. 4: 315, 1951.

Fischer, M. R., Occupation and Health, Vol. 2: 320, International Labour Off., Geneva, 1934.

Fischer, W.; in Henke, F. and Lubarsch, O., Handbuch der speziellen pathologischen Anatomie und Histologie, J. Springer, Berlin, 3, Part 3, p. 570, 1931.

Fischer-Wasels, B., Monatschr. f. Unfallh. 40: 335, 1933.

Fischer-Wasels, B., Frankfurt. Ztschr. f. Path. 49: 145, 1936.

Fitzhugh, O. G., Nelson, A. A. and Bliss, C. I., J. Pharmacol. & Exper. Therap. 80: 289, 1944.

Flory, C. N., Cancer Res. 1: 262, 1941.

Ford, C. B. and Stern, A. C., Ind. Bull. New York State Dept. of Labor 23: 253, 1944.

Foulger, J. H., Am. Ind. Hyg. Assn. Quarterly 13: 70, 1952.

Freid, T. R. and Goldberg, H., Am. J. Roentgenol. 43: 877, 1940.

Fried, B. M., Arch. of Path. 8: 46, 1929.

Fried, B. M., Med. 10: 373, 1931.

Friedell, H. L. and Rosenthal, L. M., J.A.M.A. 116: 2130, 1941.

Friedrich, G., Virchows Arch. f. path. Anat. 304: 230, 1939.

Friend, W. Z., Chem. Engin. Prog. 44: 501, 1948.

Frommel, E., Rev. d. méd., Paris 44: 31, 1927.

Fulton, J. S., Proc. Royal Soc. of Med. 42: 775, 1949.

Gaertner, K., Frankfurt. Ztschr. f. Path. 51: 89, 1937.

Gafafer, W. M. and Sitgreaves, R., Public Health Rep. 55: 1517, 1940.

Gage, C. H., American Tobacco Types, Uses and Markets, U. S. Dept. of Agric., Circular #249, Washington, D. C., 1942.

Gallie, J. F., Petroleum Refiner 23: 97, 1944.

Galloro, S., Folia Med. 21: 3, 1935.

Gardner, L. U. (See Vorwald, Pneumoconiosis).

- Gardner, L. U., J.A.M.A. 114: 535, 1940.
- Gardner, L. U., Health in Industry Transactions Bull. No. 8 of the 11th Annual Meeting of the Ind. Hyg. Foundation of America, Inc., 1946, pp. 89-94, Pittsburgh, 1947.
- Gardner, L. U. and Delahant, A. B., Am. J. Pub. Health 33: 153, 1943.
- Gärtner, H., Therap. d. Gegenwart 82: 25, 1941.
- Gärtner, H., and Brause, F. W., Med. Welt. 20: 235, 1951.
- Gelfand, M., Schistosomiasis in South Central Africa: A Clinico-Pathological Study. Publisher, Juta & Co., Ltd., for the Postgraduate Press, Cape Town, 1950, 239 pages.
- Gelman, J. G., Occupation and Health, International Labour Office, Geneva, p. 1, Suppl., 1938.
- Gerbe, H., Ztschr. f. Krebsforsch. 49: 667, 1939-1940.
- Gerrie, J., Kennedy, F. and Richardson, S. L., Canad. Med. Assn. J., Montreal 62: 544, 1950.
- Gerstel, G., Arch. f. Gewerbepath. u. Gewerbehyg. 8: 277, 1937.
- Gerstl, B., Warring, F. C., Jr. and Howlett, K. S., Jr., Am. Rev. Tuberc. 54: 470, 1946.
- Gerstl, B., Trager, M. and Szczepaniak, L. W., Proc. Soc. Exper. Biol. & Med., Utica, N. Y. 70: 697, 1949.
- Gillespie, M., Glasgow Med. J. 117: 296, 1932.
- Gillespie, M., Glasgow Med. J. 118: 26, 1932.
- Ginabat, Arch. Mal. Prof. 9: 181, 1948.
- Gloyne, S. R., Tubercle. 14: 445, 493, 550, 1933.
- Gloyne, S. R., Tubercle. 17: 5, 1935.
- Gloyne, S. R., Tubercle. 18: 100, 1936-1937.
- Gloyne, S. R., cited by Smith, W. E., Arch. Ind. Hyg. & Occup. Med. 5: 251, 1952.
- Gocher, T. E. P., Northwest Med. 40: 467, 1941.
- Goldblatt, M. W. and Wagstaff, V. A. J., J. Ind. Hyg. & Toxicol. 30:89, 1948.
- Goldblatt, M. W. and Wagstaff, V. A. J., B.I.O.S. Final Rep. 1501, Item 24, 9, 11, 17, 36, 68-71. Released for publication, 1948.

- Goldacre, R. J., Loveloss, A. and Ross, W. C. J., *Nature* 163: 667, 1949.
- Goldman, M. and Karotkin, R. H., *Am. J. Med. Sc.* 189: 400, 1935.
- Gomez, O., *Rev. Assoc. Med. Argent.* 50: 619, 1937.
- Gonnot, M. L., *Arch. Mal. Prof.* 9: 227, 1948.
- Goralewski, G., *Arch. f. Gewerbepath. u. Gewerbehyg.* 11: 108, 1941.
- Goralewski, G., *Arbeitsmedizin*, No. 26, 63 pages, Leipzig, Johann Ambrosius Barth, 1950.
- Goralewski, G. and Jaeger, R., *Arch. f. Gewerbepath. u. Gewerbehyg.* 11: 102, 1941.
- Gough, J., *J. of Path. & Bact.* 51: 277, 1940.
- Goulden, F. and Tipler, M. M., *Brit. J. Cancer* 3: 157, 1949.
- Goulden, F., Kennaway, E. L. and Urquhart, M. E., *Brit. J. of Cancer* 6: 1, 1952.
- Gowen, G. H., *Illinois Med. J.* 100: 257, 1951.
- Grace, E. J., *Am. J. Surg.* 60: 361, 1943.
- Graef, I., *Arch. of Path.* 28: 613, 1939.
- Graham, E. A., *Dis. of Chest* 18: 1, 1950.
- Graham, E. A., *Ann. Surg.* 132: 176, 1950.
- Graham, E. A., *S. Clin. North America* 30: 1259, 1950.
- Graham, E. A., *Modern Med.* 19: 99, 1951.
- Graham, E. A., *Bull. New York Academy of Med.* 27: 261, 1951.
- Graham, E. A., *New England J. of Med.*, Boston, 245: 389, 1951.
- Greenburg, L., *Monthly Review, New York State Dept. of Labor* 28: 41, 1949.
- Greenburg, L., *J.A.M.A.* 139: 815, 1949.
- Greenburg, L. and Silson, J. E., *Monthly Review. New York State Dept. of Labor* 29: 9, 1950.
- Gregorius, F., *Arch. Ind. Hyg. & Occup. Med.* 5: 196, 1952.
- Grenfell, cited by Carozzi, L., *La Medecine du Travail* 6: 1, 95, 1934.
- Grodowitz, W., *Cancer Morbidity Series, U.S.P.H.S., N.C.I.*, 46 pages, 1951.

- Gross, C. R. and Nelson, O. A., *Am. J. Public Health* 24: 36, 1934.
- Gross, E., *Klin. Wchnschr.* 15: 323, 1936.
- Gross, E., in discussion to Alwens, W., Jonas, W. and Bauke, E., *Muench. med. Wchnschr.* 83: 293, 1936.
- Gross, E., *Proc. 8th Internat. Congr. f. Unfallmed. u. Berufsk.*, Budapest, 1938. Publisher, Georg Thieme, Leipzig, 1939, p. 966.
- Gross, E., *Angew. Chem.* 53: 368, 1940.
- Gross, E. and Koelsch, F., *Arch. f. Gewerbepath. u. Gewerbehyg.* 12: 164, 1943.
- Gross, H., *Monatschr. f. Unfallh.* 38: 151, 1931.
- Gsell, O., *Schweiz. med. Wchnschr.* 81: 662, 1951.
- Gundel, M. and Heine, W., *Arch. f. Gewerbepath. u. Gewerbehyg.* 9: 248, 1938.
- Gutzeit, K., *Ztschr. f. Krebsforsch.* 19: 30, 1922.
- Haagensen, C. D., *Am. J. Cancer* 15: pt. 1, 641, 1931.
- Haerting, F. H. and Hesse, W., *Vierteljahrsschrift für Gerichtliche Medizin und Öffentliches Sanitätswesen, Neue Folge*, Berlin, 30: 102, 296, 1879.
- Haerting, F. H. and Hesse, W., *Vierteljahrsschrift für Gerichtliche Medizin und Öffentliches Sanitätswesen, Neue Folge*, Berlin, 31: 313, 1879.
- Haintz, E., *Klin. Wchnschr.* 13: 382, 1934.
- Halpert, B., *J.A.M.A.* 117: 1090, 1941.
- Hambly, A. S., Jr., *U. S. Armed Forces Med. J.* 3: 75, 1952.
- Hamilton, A., *Occupation and Health*, Internat. Labour Office, Geneva, Vol.2: 323, 1934.
- Hamlin, L. E., *J.A.M.A.* 139: 909, 1949.
- Hampeln, P., *Mitt. a. d. Grenzgeb. d. Med. u. Chir.* 36: 145, 1923.
- Hanser, R. and Simon, L., *Ztschr. f. Krebsforsch.* 51: 305, 1940-1941.
- Harding, H. E., Crout, J. L. A. and Davies, T. A. L., *Brit. J. Ind. Med.* 4: 223, 1947.
- Harding, H. E. and Oliver, G. B., *Brit. J. Ind. Med.* 6: 91, 1949.
- Hardy, H. L., *Bull. England Med. Centre* 9: 16, 1947.

- Hardy, H. L., Radiology 50: 780, 1948.
- Hardy, H. L., Lancet 2: 448, 1951.
- Hardy, H. L., Proc. Royal Soc. of Med. 44: 257, 1951.
- Hardy, H. L., Bartter, F. C. and Jaffin, A. E., Arch. Ind. Hyg. & Occup. Med. 3: 579, 1951.
- Harris, D. K., Lancet 2: 1008, 1951.
- Harris, J. H., J. Ind. Hyg. 16: 348, 1934.
- Harrison, C. V., cited by Smith, W. E., Arch. Ind. Hyg. & Occup. Med. 5: 242, 1952.
- Harrold, G. C., Meek, S. F., Collins, G. R. and Markell, T. F., J. Ind. Hyg. & Toxicol. 26: 47, 1944.
- Hasterlik, R. J., Arch. Ind. Hyg. & Occup. Med. 3: 547, 1951.
- Hayhurst, E. R., Occupation and Health, Internat. Labour Office, Geneva, Vol.1, No. 138, 1, 1930.
- Heady, J. A. and Kennaway, E. L., Brit. J. of Cancer 3: 311, 1949.
- Hegsted, D. M., McKibbin, J. M. and Drinker, C. K., Public Health Rep., Suppl. No. 179, 1945.
- Heller, I., J. Ind. Hyg. 12: 169, 1930.
- Helwig, E. B., Military Surg., Wash., D.C. 109: 540, 1951.
- Helwig, F. C., J.A.M.A. 91: 150, 1928,
- Henckel, J. F., Von der Bergsucht und Hüttenkrätze, Freiberg, 1728.
- Hendry, J. A., Rose, F. L. and Walpole, A. L., Brit. J. of Pharmacology & Chemotherapy 6: 201, 1951.
- Hendry, J. A., Homer, R. F., Rose, F. L. and Walpole, A. L., Brit. J. of Pharmacology & Chemotherapy 6: 235, 357, 1951.
- Henry, S. A., Am. J. of Cancer 31: 28, 1937.
- Henry, S. A., Cancer of the Scrotum in Relation to Occupation. Publisher, Oxford Univ. Press, London, 1946, 112 pages.
- Henry, S. A., Ann. Royal College of Surgeons of England 7: 425, 1950.
- Henschen, F., Schweiz. med. Wchnschr. 77: 968, 1947.

- Henshaw, P. S., Riley, E. F. and Stapleton, G. E., *Radiology* 49: 349, 1947.
- Hieger, I., *Brit. J. Radiol.* 20: 145, 1947.
- Higgins, H. L., *Conn. State Med. J.* 11: 330, 1947.
- Hill, A. B. and Faning, E. L., *Brit. J. Ind. Med.* 5: 1, 1948.
- Hintze, A., *Jahresk. f. arztl. Fortbild.* 7: 61, 1939.
- Hirsch, E. F. and Russell, H. B., *Arch. of Path.* 39: 281, 1945.
- Hoagland, M. B. and Hood, M. B., *Sc. Proc. Am. Assn. for Cancer Res.*, 1950.
- Hoagland, M. B., Grier, R. S. and Hood, M. B., *Cancer Res.*, Chicago 10: 629, 1950.
- Hochstatter, *Klin. Wchnschr.* 5: 1091, 1926.
- Hoffman, F. L., *Ann. Surg.* 93: 50, 1931.
- Hoffman, E., Schreus, H. I. and Zurhelle, E., *Deutsche med. Wchnschr.* 49: 633, 1923.
- Hofmann, P., *Arch. f. Gewerbepath. u. Gewerbehyg.* 7: 670, 1937.
- Holleb, H. B. and Angrist, A., *Am. J. Path.* 18: 123, 1941.
- Hollingsworth, R. K., *Ann. Int. Med.* 26: 377, 1947.
- Hollmann, W., 8th Internat. Kongr. f. Unfallmed. u. Berufsk. 2: 994, 1938.
- Holmquist, I., *Acta dermat.-venereol.* 31, Suppl. 26: 214, 1951.
- Homburger, F., *Am. J. Path.* 19: 797, 1943.
- Hopkins, R. and van Studdiford, M. T., *Arch. f. Dermat. u. Syph.* 29: 408, 1929.
- Horning, R., *Ztschr. f. Krebsforsch.* 47: 281, 1938.
- Hruby, A. J. and Sweany, H. C., *Arch. Int. Med.* 52: 497, 1933.
- Hübner, Otto, *Arch. f. Gewerbepath. u. Gewerbehyg.* 9: 426, 1938-1939.
- Hueck, W., *Ztschr. f. Krebsforsch.* 49: 312, 1939.
- Hueper, W. C., *Occupational Tumors and Allied Discases*, Publisher, C. C. Thomas, Springfield, Ill., 1942, 890 pages.
- Hueper, W. C., *Public Health Rep.*, Suppl. 209, 1948, 69 pages.
- Hueper, W. C., *Occup. Med.* 5: 157, 1948.

- Hueper, W. C., Southern Med. J. 43: 118, 1950.
- Hueper, W. C., Public Health Technical Monograph No. 1, U.S.P.H.S., 1950,
37 pages.
- Hueper, W. C., Am. J. of Med. 8: 355, 1950.
- Hueper, W. C., Arch. Ind. Hyg. & Occup. Med. 2: 325, 1950.
- Hueper, W. C., Ind. Med. & Surg. 20: 49, 1951.
- Hueper, W. C., Arch. Ind. Hyg. & Occup. Med. 5: 288, 1952.
- Hueper, W. C., Ind. Med. & Surg. 21: 71, 1952.
- Hueper, W. C., Public Health Rep. 67: 773, 1952.
- Hueper, W. C., Texas Reports on Biol. & Med. 10: 167, 1952.
- Hueper, W. C. and Mancuso, T. F., Publ. Health Rep. 67: 644, 1952.
- Hueper, W. C., Zuefle, J. H., Link, A. M. and Johnson, M. G. Published in
J. Nat. Cancer Inst. 13: 291, 1952.
- Hugounenq, L., Ann. Hyg. publ., N.s. 17: 1, 1939.
- Huguenin, R., Fauvet, J. and Bourdin, J., Bull. et Memoires de la
Société des Hopitaux de Paris 65: 1020, 4th Series, 1949.
- Humperdinck, K., Deutsche med. Wchnschr. 16: 68, 1942.
- Humphris, F. H., Lancet 1: 597, 1934.
- Hünemann, T., Ztschr. f. Laryngol. 17: 369, 1929.
- Hunter, D., Milton, R., Perry, K. M. A. and Thompson, D. R., Brit. J.
Ind. Med. 1: 159, 1944.
- Husted, E. and Biilmann, G., Hospitalstidende 79: 325, 1935.
- Husted, E. and Biilmann, G., Acta path. et. microbiol. Scandinav. 14:
141, 1937.
- Hyslop, F., Palmes, E. D., Alford, W. C., Monaco, A. R. and Fairhall,
L. T., Nat. Inst. Health Bull. 181: 47, 1943.
- Ichok, G., Ann. Hyg. publ. ind. sociale 7: 113, 1929.
- Ikeda, K., Am. J. Clin. Path. 5: 89, 1935.
- Imprescia, S., Proc. 1st Internat. Congr. Chest Diseases, Rome, 1950.

- Ingalls, T. H., Arch. Ind. Hyg. & Occup. Med. 1: 662, 1950.
- Internat. Nickel Co., Canadian Mining J. 67: 315, 370, 457, 523, 534, 552, 553, 1946.
- Jackson, C. and Jackson, C. L., Arch. Otolaryng. 33: 45, 1941.
- Jaeger, R. and Jaeger, F., Arch. f. Gewerbepath. u. Gewerbehyg. 11: 117, 1941.
- Jaffe, F. A., Am. J. of Path. 27: 909, 1951.
- Jaffe, R. H., J. Lab. & Clin. Med. 20: 1227, 1935.
- Jephcott, C. M. and Johnston, J. H., Arch. Ind. Hyg. & Occup. Med. 1: 323, 1950.
- Jetter, W. W., Am. J. Path. 24: 690, 1948.
- Joannovic, Georg, Klin. Wehnschr. 2: 2301, 1923.
- Johnstone, R. T., J.A.M.A. 114: 546, 1940.
- Jones, T. R. and Lockhart, J. A., Texas St. J. Med. 39: 532, 1944.
- Jöten, K. W. and Reploh, H., Rassegna di med. indust. 9: 395, 1938.
- Jöten, K. W., Reploh, H. and Hagemann, G., Arch f. Gewerbepath. u. Gewerbehyg. 9: 314, 1939.
- Kahlau, G., Frankfurt. Ztschr. f. Path. 55: 364, 1941.
- Kahlau, G., Verhandl. d. deutsch. path. Gesellsch. 32: 377, 1950.
- Kahlstorf, A., Deutsche Arch. f. klin. Med. 184: 466, 1939.
- Kalbfleisch, H. H., Arch. f. Gewerbepath. u. Gewerbehyg. 7: 699, 1936.
- Kalbfleisch, H. H., Smmalg. f. Vergiftungsf. 8: 27, 1937.
- Kaplan, L., Am. J. Dis. Children 62: 1217, 1941.
- Katz, K., Ztschr. f. Krebsforsch. 25: 368, 1927.
- Kawahata, K., Gann 30: 341, 1936.
- Kazanjan, V. H. and Joseph, A. T., Plastic and Reconstructive Surg. 6: 156, 1950.
- Kennaway, E. L., J. Ind. Hyg. 5: 462, 1924.
- Kennaway, E. L., J. Ind. Hyg. 7: 69, 1925.
- Kennaway, E. L., Brit. Med. J. 2: 1, 1925.

- Kennaway, E. L., Brit. J. of Cancer 4: 158, 1950.
- Kennaway, E. L., Cancer 4: 638, 1951.
- Kennaway, E. L. and Kennaway, N. M., J. Hyg. 36: 236, 1936.
- Kennaway, E. L. and Kennaway, N. M., Brit. J. Cancer 1: 260, 1947.
- Kennaway, E. L. and Kennaway, N. M., Brit. J. Cancer 5: 153, 1951.
- Kennaway, J., Brit. Med. J. 1: 22, 1948.
- Kennedy, B. J., Pare, J. A. P., Pump, K. K., Beck, J. C., Johnson, L. G.,
Epstein, N. B., Venning, E. H. and Browne, J. S. L., Am. J. Med.
10: 134, 1951.
- Kikuth, Virchows Arch. f. path. Anat. 255: 107, 1925.
- Klemperer, F., Arch. Ind. Hyg. & Occup. Med. 3: 625, 1951.
- Klemperer, F. W., Martin, A. P. and Van Riper, J., Arch. Ind. Hyg. &
Occup. Med. 4: 251, 1951.
- Kline, E. M., Inkley, S. R. and Pritchard, W. H., Arch. Ind. Hyg. &
Occup. Med. 3: 549, 1951.
- Kling, A., Samssonow, N., and Heres, M., Bull. Acad. de med., Paris 119:
439, 1938. Quoted in Paris Letter, J.A.M.A. 113: 245, 1939.
- Klotz, M. O., Canad. Med. Assn. J. 17: 989, 1927.
- Klotz, M. O., Am. J. Cancer 35: 38, 1939.
- Klotz, M. O. and Simpson, W., Emanuel Libman Anniv. Vol.2: 685, 1932.
- Koelsch, F., J. Ind. Hyg. 5: 87, 1923.
- Koelsch, F., Theophrastus von Hohenheim, genannt Paracelsus: Von der
Bergsucht und Anderen Bergkrankheiten, Berlin, 1925.
- Koelsch, F., Handbuch der Berufskrankheiten, Vol.1, Gustav Fischer,
Jena, p. 621, 1935.
- Koelsch, F., Acta Unio internat. contra cancerum 3: 243, 1938.
- Koelsch, F., Arch. f. Gewerbepath. u. Gewerbehyg. 10: 519, 1940-1941.
- Koelsch, F., Beitr. z. Klin. d. Tuberk. 97: 688, 1942.
- Kolzoff, H., Zentralbl. f. Gewerbehyg. 3: 339, 1927.
- Konrad, A. and Franke, W., Deutsche med. Wchnschr. 55: 652, 1929.
- Konwaler, B. E. and Reingold, I. M., Cancer 5: 525, 1952.

- Koppisch, E., J.A.M.A. 121: 936, 1943.
- Kosir, A., Oncologia 4: 109, 1951.
- Kotzing, K., Arch. f. Gewerbepath. u. Gewerbehyg. 4: 500, 1932-1933.
- Kouwenaar, W., J. Nat. Cancer Inst. 11: 640, 1950.
- Kouwenaar, W., Documenta Neerlandica et Indonesica de Morbis Tropicis 3: 357, 1951.
- Krafft, K., Ber. 8th Internat. Kongr. f. Unfallmed. u. Berufsk. 2: 1054, 1939.
- Krafft, K., Zentr. Gewerbehyg. Unfallkerhiit. 27: 122, 1940.
- Kraft, I. A., Ztschr. f. Krebsforsch. 42: 51, 1934.
- Kreyberg, L., Tidsskr. F. D. Norske Laegeforen 70: 682, 1950.
- Kreyberg, L., Brit. J. of Cancer 6: 112, 1952.
- Krueger, E., Rostoski, O. and Saupe, E., Ztschr. f. klin. Med. 107: 365, 1928.
- Kruger, E., Rostoski, O. and Saupe, E., Arch. f. Gewerbepath. u. Gewerbehyg. 2: 558, 1931.
- Kuroda, S. and Kawahata, K., Ztschr. f. Krebsforsch. 45: 36, 1936.
- Kuroda, S. and Kawahata, K., Jap. J. M. Sc., 8, Int. Med. Pediat. & Psychiat. 5: 41, 1938.
- Lacassagne, A., Actualités Scientifiques Et Industrielles, Chap. 4, p. 55, Editors, Hermann Et C^{ie}, Paris, 1945.
- Landis, Funk, Smyth and Miller, cited by Allevi.
- Lange, K., Ztschr. f. Krebsforsch. 42: 306, 1935.
- Lanza, A. J., J. Missouri State Med. Assn. 42: 765, 1945.
- Lanza, A. J., Proc. 42nd Ann. Conf. of Air Pollution and Smoke Prevention Assn. of America, Birmingham, Ala., 1949.
- Lanza, A. J., Safety Engineering, April 1950, 4 pages.
- Large, H. L., Jr. and Stumpe, A. R., So. Med. J., Birmingham, Ala., 44: 36, 1951.
- Larson, C. P., Northwest Med. 37: 183, 1938.
- Laskin, S., Turner, R. A. N. and Stokinger, H. E., U.S. Atomic Energy Comm. MDDC-1355, 1947.
- Leach, J. E., Farrow, J. H., Foote, F. W. and Wawro, N.W., Am. J. Roentgenol. 47: 740, 1942.
- Lecoeur, J., Presse méd. 50: 415, 1942.
- Legge, T. M., Brit. Med. J. 2: 1100, 1922.

- Lehmann, K. B.; Arch. f. Hyg. 28: 291, 1897.
- Lehmann, K. B., Schriften aus dem Gesamtgebiet der Gewerbehygiene 2: 1, 1914.
- Lehmann, K. B., Arch. f. Hyg. 104: 105, 1930.
- Lehmann, K. B., Zentralbl. f. Gewerbehyg. 9: 168, 1932.
- Leitch, A., Brit. Med. J. 2: 941, 1924.
- Leitch, Sternberg, cited by Willis and Brutsaert.
- Leiter, J. and Shear, M. J., J. Nat. Cancer Inst. 3: 167, 1942.
- Leiter, J., Shinkin, M. B. and Shear, M. J., J. Nat. Cancer Inst. 3: 155, 1942.
- Leroux-Robert, Bull. Acad. de. med., Paris 116: 875, 1936.
- Letterer, E., Arch. f. Gewerbepath. u. Gewerbehyg. 9: 496, 1938-1939.
- Letterer, E., Neidhardt and Klett, H., Arch. f. Gewerbepath. u. Gewerbehyg. 12: 323, 1944.
- Levin, M. L., Goldstein, H. and Gerhardt, P. R., J.A.M.A. 143: 36, 1950.
- Levin, M., Goldstein, H. and Gerhardt, P. R., A Preliminary Report. Bu. of Cancer Control, New York State Dept. of Health, Dec. 5, 1949; J.A.M.A. 143: 336, 1950.
- Lickint, F., Ztschr. f. Krebsforsch. 30: 349, 1929-1930.
- Lickint, F., Muench. med. Wchnschr. 82: 1232, 1935.
- Lieberman, H., New Eng. J. Med. 225: 132, 1941.
- Lillie, R. D. and Smith, M. I., Am. J. Path. 16: 223, 1940.
- Lindemann, Deutsche med. Presse 20: 3, 1916.
- Linzbach, A. J. and Wedler, H. W., Virchows Arch. f. path. Anat. 307: 387, 1941.
- Lisco, H. and Finkel, M. P., Fed. Proc. 8: 360, 1949.
- Ljvraga, P., Pathologica 26: 726, 1934.
- Løken, A. C., Tidsskr. Norske Lægefor. 70: 376, 1950.
- Long, C. F., Ind. Med. 8: 365, 1939.
- Lorentz, F. H., Die med. Welt. 4: 200, 1930.
- Lorenz, E., J. Nat. Cancer Inst. 5: 1, 1944.
- Lorenz, E., Stewart, H. L., Daniels, J. H. and Nelson, C. V., Cancer Res. 3: 123, 1943.

Lorenz, E., Heston, W. E., Deringer, M. K. and Eschenbrenner, J. B., J. Nat. Cancer Inst. 6: 349, 1946.

Lorenz, E., Heston, W. E., Eschenbrenner, J. B. and Deringer, M. K., Radiology 49: 274, 1947.

Love, R. A., U. S. Atomic Energy Comm., AECU-1620, Brookhaven Nat. Lab., Tech. Inf. Serv., Oak Ridge, Tenn. Issued, 1951.

Lowry, P. H., Abstract, Arch. Ind. Hyg. & Occup. Med. 4: 617, 1951.

Lowy, J., Med. Klin. 24: 1784, 1928.

Lowy, J., Med. Klin. 25: 141, 1929.

Lowy, J., Med. Klin. 18: 619, 1936.

Lowy, J., Acta Unio internat. contra cancerum 3: 182, 1938.

Lowy, J., Occupation and Health, Internat. Labour Office, Suppl., Geneva, p. 1, 1939.

Luckow, Ztschr. f. arztl. Fortbild. 30: 702, 1933.

Ludwig, P. and Lorensen, E., Strahlenther. 17: 428, 1924.

Lü-Fu-hua, Frankfurt. Ztschr. f. Path. 46: 513, 1933-1934.

Lü-Fu-hua, Frankfurt. Ztschr. f. Path. 47: 52, 1934-1935.

Lukanin, W. P., Arch. of Hyg. 104: 166, 1930.

Lynch, K. M., J.A.M.A. 109: 1974, 1937.

Lynch, K. M. and Carmon, W. M., Dis. of Chest 14: 874, 1948.

Lynch, K. M. and Smith, W. A., Am. J. Cancer 24: 56, 1935.

Lynch, K. M. and Smith, W. A., Am. J. Cancer 36: 567, 1939.

Machle, W., Radiology 50: 755, 1948.

Machle, W. and Gregorius, F., Proc. 9th Internat. Congr. on Ind. Med., London, p. 454, 1948.

Machle, W. and Gregorius, F., Public Health Rep. 63: 1114, 1948.

Machle, W., Reyor, E. C. and Tedbrook, H., Proc. 9th Internat. Congr. on Ind. Med., London, p. 615, 1948.

Macklin, M. T. and Macklin, C. C., Arch. of Path. 30: 924, 1940.

Macklin, M. T., Ann. Intern. Med. 17: 308, 1942.

- MacMahon, H. E., *Am. J. Path.* 28: 531, 1952.
- Mainzer, F., *Acta med. Scand.* 85: 538, 1935.
- Maisin, J., *Bruxelles méd.* 14: 544, 1934.
- Manas, M. A., *Revista Mexicana de Tuberculosis, Mexico, D. F.* 7: 391, 1945.
- Mancioli, G., *Rass. Med. Industriale* 15: 170, 1950.
- Mancuso, T. F., *Ind. Med. & Surg.* 20: 393, 1951.
- Mancuso, T. F., personal communication.
- Mancuso and Urone, personal communication.
- Mancuso, T. F. and Hueper, W. C., *Ind. Med. & Surg.* 20: 358, 1951.
- Mangelsdorff, B., *Fortschr. a. d. Geb. d. Rontenstrahlen.* 74: 336, 1951.
- Manville, T. A., *Am. J. Public Health* 29: 709, 1939.
- Marcus, S. C., *Cancer Morbidity Series 4, U.S.P.H.S., N.C.I.*, 1951, 39 pages.
- Marie-Améro, M., *Bull. soc. sci. hyg. aliment.* 18: 42, 1930.
- Martin, H., *Cancer of the Head and Neck, R. R. Donnelley & Sons, Co., Chicago, Ill.*, p. 9, 1949.
- Martineck, *Arbeit und Gesundheit, Heft 29, Dritte Verordnung der Unfallversicherung auf Berufskrankheiten, von 1936. Publisher Georg Thieme, Leipzig, 1937.*
- Martland, H. S., *Am. J. Cancer* 15: 2435, 1931.
- Martland, H. S., *Textbook of Med. (Cecil)*, 7th Edition: 582, 1947.
- Martland, H. S., Brodtkin, H. A. and Martland, H. S., Jr., *N. J. Med. Soc. J.*, Trenton, 45: 5, 1948.
- Marx, W., *Tuberkulosearzt* 4: 102, 1950.
- Mason, G. A., *Lancet* 2: 587, 1949.
- Matz, P. B., *J.A.M.A.* 111: 2086, 1938.
- Maxwell, I., *M. J. Australia* 2: 168, 1934.
- Mayers, M. R., *New York State Dept. of Labor. Monthly Review. Div. of Ind. Hyg. & Safety Standards*, 30: 13, 1951.
- McCord, C. P., *Ind. Med. & Surg.* 19: 97, 1950.
- McCord, C. P. and Friedlander, A., *Arch. Int. Med.* 37: 641, 1926.

- McCormick, W. E., Smith, M. and Marsh, S. P., J. Ind. Hyg. & Toxicol. 30: 43, 1948.
- McCrae, T., Funk, E. H. and Jackson, C., J.A.M.A. 89: 1140, 1927.
- McDonald, S. and Woodhouse, D. L., J. Path. & Bact., Edinburgh 54: 1, 1942.
- McIntosh, H. C. and Spitz, S., Am. J. Roentgenol. 41: 605, 1939.
- McLaughlin, A. I. G., Industrial Lung Diseases of Iron and Steel Foundry Workers, Great Brit. Factory Dept., Ministry of Labour & National Service, H. M. Stationery Office, London, 1950.
- McNally, W. D., Am. J. Cancer 16: 1502, 1932.
- Medicolegal Abstract, J.A.M.A. 147: 778, 1951.
- Menz, H., Schweiz. med. Wchnschr. 77: 895, 1947.
- Merewether, E. R., Ann. Rep. of the Chief Insp. of Factories, H. M. Stationery Office, 1947, p. 15.
- Merewether, E. R., Ann. Rep. of the Chief Insp. of Factories and Workshops, 1948. H. M. Stationery Office, London, 1949.
- Mertens, V. E., Ztschr. f. Krebsforsch. 32: 82, 1930.
- Mertens, V. E., Ztschr. f. Krebsforsch. 51: 183, 1940-1941.
- Metropolitan Life Insurance Co., 1937-1939, Mortality Experience Among White Male Wage Earners Insured Under Industrial Policies.
- Michalowsky, I., Virchows Arch. f. path. Anat. 267: 27, 1928.
- Middleton, E. L., Lancet 2: 1, 59, 1936.
- Mills, C. A., Cincinnati J. Med. 27: 692, 1946.
- Mills, C. A. and Porter, M. M., Cancer Res. 10: 539, 1950.
- Mödder, H. and Schmitt, T., Deutsche med. Wchnschr. 76: 84, 1951.
- Montgomery, H. and Waisman, M., J. of Invest. Dermat. 4: 365, 1941.
- Moore, R. M. and Neal, M. P., So. Med. J. 30: 395, 1937.
- Moore, R. M. and Schmoisser, H. C., Am. Rev. Tuberc. 35: 336, 1937.
- Morgan, personal communication.
- Morgis, G. G. and Forbes, J. J., Bu. of Mines Information Circular 7574. U. S. Dept. of Interior, 1950.
- Mote, R. H., Arsenic, in Pehrson, E. W. and Matthews, A. I., Mineral Year Book, 1946, Dept. of Interior, Wash., D. C., 1948, p. 137.

- Motley, H. L., Ellis, M. M. and Ellis, M. D., J.A.M.A. 109: 1718, 1937.
- Mott, F. W., Arch. Neurol. Path. Lab., London County Asylum 3: 246, 1907.
- Moxon, A. L. and Rhian, M., Physiol. Rev. 23: 305, 1943.
- Mueller, F. H., Ztschr. f. Krebsforsch. 49: 57, 1939.
- Müllschitzky, A., Dermat. Wehnschr. 109: 973, 1939.
- Müllschitzky, A., Wien. med. Klin. 35: 1401, 1939.
- Muntean, E. and Amon, R., Fortschr. a. d. Geb. d. Röntgenstrahlen. 73: 156, 1950.
- Nash, P., Lancet 1: 519, 1950.
- Nash, P., Grier, R. S. and Freiman, D. G., Proc. 9th Internat. Congr. on Ind. Med., London, p. 366, 1948.
- Neitzel, E., Arbeitsschutz., No. 1, Johann Ambrosius Barth, Leipzig, p. 46, 1935.
- Neitzel, E., Arbeitsschutz., No. 3, Johann Ambrosius Barth, Leipzig, p. 70, 1937.
- Nelson, A. A., Fitzhugh, O. G. and Calvery, H. O., Cancer Res. 3: 230, 1943.
- Neubauer, O., Brit. J. Cancer 1: 192, 1947.
- Newman, D. A., Glasgow Med. J. 33: 469, 1890.
- Nicoloff, P., Verhandl. d. 6th Internat. Congr. f. Berufskrank., p. 669, 1931.
- Nieberle, K., Ztschr. f. Krebsforsch. 49: 137, 1939-1940.
- Nordmann, M., Ztschr. f. Krebsforsch. 47: 288, 1938.
- Nordmann, M., Ber. 8th Internat. Kongr. f. Unfallmed. u. Berufsk., Leipzig, Georg Thieme, 2: 983, 1939.
- Nordmann, M. and Sorge, A., Ztschr. f. Krebsforsch. 51: 168, 1940-1941.
- Nuck, Remy, E. and Holtzmann, F., Ztschr. f. Hyg. 109: 598, 1929.
- Ochsner, A. and De Bakey, M., Arch. Surg. 42: 209, 1941.
- Ochsner, A., Dixon, J. L. and De Bakey, M., Dis. of Chest 11: 3, 1945.
- Ochsner, A., De Bakey, M., et al., Chicago Med. Soc. Bull. 51: 127, 1948.
- Ochsner, A., De Camp, P. T., De Bakey, M. E. and Ray, C. J., J.A.M.A. 148: 691, 1952.
- Odier, E., Neoplasmes 4: 145, 1925.
- O'Donovan, W. J., Brit. J. Dermat. & Syph. 32: 215, 1920.

- O'Donovan, W. J., Brit. Med. J. 2: 292, 1932.
- Oldofredi, F. J., Krebsarzt. 3: 86, 1948.
- Oliver, J. H., Lancet 2: 266, 1951.
- Ollivier, H., Morand, P. and Brun, R., Arch. d. mal. Profess. 10: 516, 1949.
- Olson, K. B., Am. J. Path. 11: 449, 1935.
- Olson, K., J.A.M.A. 105: 802, 1935.
- Oppenheimer, B. S., Oppenheimer, E. T. and Stout, A. P., Proc. Soc. for Exper. Biol. & Med. 67: 33, 1948.
- Oppenheimer, B. S., Oppenheimer, E. T. and Stout, A. P., Proc. Soc. for Exper. Biol. & Med. 79: 366, 1952.
- Owen, T. K., Brit. J. of Cancer 5: 382, 1951.
- Palitsch, F., Zentralbl. f. Gewerbehyg. 9: 225, 1921.
- Pancoast, H. K., Miller, T. G. and Landis, H. R., Trans. Assn. Am. Phys. 32: 97, 1917.
- Pancoast, H. K. and Pendergrass, E. P., J.A.M.A. 101: 587, 1933.
- Parkes, G. D., Mellor's Modern Inorganic Chemistry, Longmans, Green & Co., New York, 1939, p. 684.
- Pascucci, L. M., Radiology 50: 23, 1948.
- Passey, R. D., Brit. Med. J. 2: 1112, 1922.
- Passey, R. D. and Holmes, J. McD., Quart. J. Med. 4: 321, 1935.
- Pearce, G. W., Mattson, A. M. and Hayes, W. J., Science 116: 254, 1952.
- Peller, S., Human Biol. 11: 130, 1939.
- Pendergrass, E. P. and Leopold, S. S., J.A.M.A. 127: 701, 1945.
- Pendergrass, E. P. and Pryde, A. M., J. Ind. Hyg. & Toxicol. 30: 119, 1948.
- Pendergrass, E. P., Lane, E. L. and Ostrum, H. W., Am. J. Roentgenol. & Radium Therapy 61: 443, 1949.
- Perret, New Orleans Med. J. 80: 213, 1927.
- Perrone, J. A. and Levinson, J. P., Ann. Int. Med. 17: 11, 1942.
- Perry, K. M. A., Thorax, London 2: 91, 1947.
- Perry, K., Bowler, R. G., Buckell, H. M., Druett, H. A. and Schilling, R. S. F., Brit. J. Ind. Med. 1: 6, 1948.

- Pessano, J. E., *Semana med.*, 2: 210, 1942.
- Pfeil, E., *Deutsche med. Wchnschr.* 61: 1197, 1935.
- Philips, F. S., *J. Pharmacol. & Exper. Therap.* 99: 281, 1950.
- Pilgersdorfer, W., *Wien. Arch. f. inn. Med.* 30: 71, 1937.
- Pirchan, A. and Sikl, H., *Am. J. Cancer* 16: 681, 1932.
- Plazy, Gouriou and Germain, *Bull. et mem. Soc. med. d. hop. de Paris* 55: 1100, 1931.
- Policard, A., *J. Ind. Hyg. & Toxicol.* 30: 105, 1948.
- Pomeranz, R. and Brodtkin, H. A., *J. Inter. College of Surgeons, Chicago* 15: 633, 1951.
- Popp, H., *Ztschr. f. Angew Chem.* 41: 838, 1928.
- Pott, P., *Chirurgical Observations Relative to the Cataract, the Polypus of the Nose, the Cancer of the Scrotum, the Different Kinds of Ruptures, and the Mortification of the Toes and Feet.* Printed by T. J. Carnogie, 1775, p. 63, Hower, Clark & Pollins, London.
- Potter, E. A., *Cancer Res.* 7: 351, 1947.
- Prell, H., *Arch. f. Gewerbepath. u. Gewerbehyg.* 7: 656, 1937.
- Probst, R., *Ztschr. f. Krebsforsch.* 25: 431, 1927.
- Proc. First National Cancer Conference, p. 189, 1949, American Cancer Society and The National Cancer Institute.
- Pyre, J. and Oatway, W. H., Jr., *Ariz. Med., Phoenix* 4: 21, 1947.
- Query, J.A.M.A. 103: 280, 1934.
- Query, J.A.M.A. 141: 574, 1949.
- Rajewsky, B., *Ztschr. f. Krebsforsch.* 49: 315, 1939-1940.
- Rajewsky, B., Schraub, A. and Kahlau, G., *Naturwissenschaften* 31: 170, 1943.
- Ray, S. C., King, E. J. and Harrison, C. V., *Brit. J. Ind. Med., London* 8: 68, 1951.
- Registrar-General, *Decennial Suppl., England and Wales, 1921, London, H. M. Stationery Office, 1927; Decennial Suppl., England and Wales, 1931, London, 1938.*
- Rehner, J., Jr., *J. Am. Chem. Society* 62: 2243, 1940.
- Remington, R. E., *J. Am. Chem. Society* 49: 1910, 1927.
- Reynolds, P. W., *Arch. Ind. Hyg. & Occup. Med.* 3: 575, 1951.

- Rice, C. M., J. Lab. & Clin. Med. 21: 906, 1936.
- Rigdon, R. H. and Kirchoff, H., Texas Rep. on Biol. & Med. 10: 76, 1952.
- Risel, cited by Thiele, A., Die Schneeberger Lungenkrankheit, in Fabrikärzte der chemischen Industrie. Merkblätter ueber berufliche Erkrankungen. J. Springer, Berlin, 1930, 3rd edition.
- Rizzuti, A. B., New York State J. of Med. 51: 1065, 1951.
- Robbins, E. and Silverman, G., Cancer 2: 65, 1949.
- Rodenacker, cited by Hueper.
- Roesch, H., Virchows Arch. f. path. Anat. 245: 1, 1923.
- Roffo, A. H., Ztschr. f. Krebsforsch. 33: 321, 1930-1931.
- Roffo, A. H., Bol. Inst. de med. exper. para el estud. y trat. d. cancer 8: 545, 1931.
- Roffo, A. H., Am. J. Cancer 18: 136, 1933.
- Roffo, A. H., Bol. Inst. de med. exper. para el estud. y trat. d. cancer 14: 311, 1937.
- Roffo, A. H., Deutsche med. Wchnschr. 63: 1267, 1937.
- Roffo, A. H., Acta Unio internat. contra cancrum 4: 755, 1939.
- Roffo, A. H., Deutsche med. Wchnschr. 65: 963, 1939.
- Roffo, A. H., Bol. Inst. de med. exper. para el estud. y trat. d. cancer 16: 297, 1939.
- Roffo, A. H., Ztschr. f. Krebsforsch. 49: 588, 1939-1940.
- Roffo, A. H., Monatschr. f. Krebsbekampf. 8: 97, 1940.
- Roffo, A. H., Bol. Inst. de med. exper. para el estud. y trat. d. cancer 17: 699, 1940.
- Roffo, A. H., Schweiz. med. Wchnschr. 71: 549, 1941.
- Roffo, A. H., Bol. Inst. de med. exper. para el estud. y trat. d. cancer 19: 431, 1942.
- Roffo, A. H., Bol. Inst. de med. exper. para el estud. y trat. d. cancer 20: 189, 1943.
- Roffo, A. H., Rev. Liga Puerturriq. Contra El Cancer 3: 122, 1943.
- Roffo, A. H., Rev. Med. De Cir. Ginec. y Cancer 11: 259, 1943.
- Roffo, A. H., Bol. Inst. de med. exper. para el estud. y trat. d. cancer 20: 103, 1943.
- Roffo, A. H., La Prensa Med. Argentina 31: 688, 1944.

- Rogers, W. L., Arch. Int. Med. 49: 1058, 1932.
- Rosahn, P. D., Review of Yale Autopsy Protocols, 1917 to 1937. Arch. Path. 29: 649, 1940.
- Rosedale, R. S. and McKay, D. R., Am. J. Cancer 26: 493, 1936.
- Rospide, P. C., Prensa Medica Argentina, Buenos Aires 35: 2508, 1948.
- Ross, P., Brit. Med. J. 2: 369, 1948.
- Rossier, P. H. and Buhlmann, A., Schweiz. med. Wchschr. 79: 685, 1949.
- Rossle, R., Ztschr. f. Krebsforsch. 32: 686, 1930.
- Rostoski, O., Internat. Cancer Conf., London, 1928, p. 269.
- Rostoski, O., Saupe, E. and Schmorl, G., Ztschr. f. Krebsforsch. 23: 360, 1926.
- Rostoski, O. and Saupe, E., Arch. f. Gewerbepath. u. Gewerbehyg. 1: 731, 1930.
- Rottmann, Inaug. Diss., Wurzburg, 1898.
- Rousseau, L., Semaine d. hop. de Paris 23: 1811, 1947.
- Royer, P., Biologie Médicale. 41: 149, 1952.
- Russell, B. F. and Klaber, R., Proc. Roy. Soc. Med. 38: 128, 1945.
- Rüttner, J. R., Oncologia 2: 115, 1949.
- Saita, G. I., Med. d. Lavoro 39: 105, 1948.
- Sander, O. A., Am. J. Roentgenol. & Rad. Therap. 58: 277, 1947.
- Sander, O. A., Arch. Ind. Hyg. & Occup. Med. 3: 565, 1951.
- Sante, L. R., Am. J. Roentgenol. 62: 788, 1949.
- Sappington, C. O., Essentials of Industrial Health. Publisher, J. B. Lippincott Co., Philadelphia, Pa., 1943, pp. 218, 236.
- Saupe, E., Ztschr. f. Krebsforsch. 32: 687, 1930.
- Saupe, E., Arch. f. Gewerbepath. u. Gewerbehyg. 1: 582, 1930.
- Saupe, E., Zentralbl. f. inn. Med. 54: 825, 1933.
- Saupe, E., Fortschr. a. d. Geb. d. Röntgenstrahlen, Ergänzungsbande. 60: 163, 1939.
- Schachter, M., J. de med. de Paris 52: 700, 1932.
- Schairer, E. and Schöninger, E., Ztschr. f. Krebsforsch. 54: 261, 1944.
- Schanberg, J. F., J. Cut. Dis. 28: 644, 1910.
- Scheffler, C. L., Die Gesundheit der Bergleute. Chemnitz, 1770.

Schildknecht, C. E., Vinyl and Related Polymers. Publisher, J. Wiley & Sons, Inc., New York, 1952, p. 539.

Schinz, H. R., Schweiz. med. Wchnschr. 72: 1070, 1942.

Schinz, H. R., Rosin, S. and Senti, A., Bulletin der Schweizerischen Akademie der Medizinischen Wissenschaften 2, p. 131, 1946.

Schiøtz, E. H., Proc. 9th Internat. Congr. on Ind. Med., London, 798, 1948.

Schittenhelm, A. and Stockinger, W., Ztschr. f. exper. Med. 45: 58, 1925.

Schmidtman, M., Ztschr. f. Krebsforsch. 32: 677, 1930.

Schmidtman, M., Arch. f. Gewerbepath. u. Gewerbehyg. 8: 1, 1937.

Schmorl, G., Verhandl. d. deutsch. path. Gesellsch. 19: 192, 1923.

Schmorl, G., Rep. Internat. Cancer Conf., London, 1928, p. 272, Publisher, John Wright & Sons, Ltd., Bristol.

Schneider, L., New Eng. J. Med. 240: 284, 1949.

Schnurer, L., J. Ind. Hyg. & Toxicol. 20: 14, 1938.

Schnurer, L. and Haythorn, S., Am. J. Path. 13: 676, 799, 1937.

Schoenher, Ztschr. f. Krebsforsch. 27: 436, 1928.

Schopper, W., Arch. f. Hyg. 104: 175, 1930.

Schrapf, R., Le Médecin D'Usine, Paris 10: 137, 1948.

Schrek, R., Baker, L. A., Ballard, G. P. and Dolgoff, S., Cancer Res. 10: 49, 1950.

Schulte, G., Fortschr. a. d. Geb. d. Röntgenstrahlen. 41: 444, 1930.

Schulz, O., Arch. f. Gewerbepath. u. Gewerbehyg. 6: 117, 1935.

Schurch, O. and Winterstein, A., Ztschr. f. Krebsforsch. 42: 76, 1935.

Schurch, O. and Winterstein, A., Ztschr. f. Physiol. Chem. 236: 79, 1935.

Schurch, O. and Winterstein, A., Ztschr. f. Krebsforsch. 46: 414, 1937.

Schwartz, L. and Tulipan, L., Occupational Diseases of the Skin. Publisher, Lea & Febiger, Philadelphia, Pa., 132 pages, 1939.

Schwartz, L. and Dunn, J. E., Ind. Med. 11: 432, 1942.

Schwartz, P., Beitr. z. Klin. d. Tuberk. 103: 192, 1950.

Schwellnus, H. and Kleinsorg, H., Deutsche Ztschr. f. d. ges. gerichtl. Med., Heidelberg 39: 577, 1949.

- Schwyster, M., Frankfurt, Ztschr. f. Path. 36: 146, 1928.
- Scott, A., Brit. Med. J. 2: 281, 1922.
- Scott, A., Brit. Med. J. 2: 1108, 1922.
- Seelig, M. G. and Cooper, Z. K., Am. J. Cancer 17: 589, 1933.
- Seelig, M. G. and Benignus, E. L., Am. J. Cancer 28: 96, 1936.
- Seelig, M. G. and Benignus, E. L., Am. J. Cancer 33: 549, 1938.
- Seelig, M. G. and Benignus, E. L., Am. J. Cancer 34: 391, 1938.
- Seifter, J., Ehrich, W. E., Hudyma, G. and Mueller, G., Science 103: 762, 1946.
- Seyfarth, C., Deutsche med. Wchnschr. 50: 1497, 1924.
- Sharrah, J. S., Pennsylvania State Dept. of Health, Harrisburg, Div. of Air Pollution, 1951, 15 pages.
- Shaver, C. G., Radiology 50: 760, 1948.
- Shaver, C. G. and Riddell, A. R., J. Ind. Hyg. & Toxicol. 29: 145, 1947.
- Shaw, L. F. B. and Ghazeeb, A. N., J. Path. & Bact. 46: 401, 1938.
- Shennan, R., J. Path. & Bact. 31: 365, 1928.
- Shilen, J., Mellor, J. F., Jr., Koppenhaver, F. B., Cleland, J. G., Galloway, A.E. and Lutz, L. R., Ind. Med. 18: 109, 1949.
- Shimkin, M. H. and Lieter, J., J. Nat. Cancer Inst. 1: 241, 1940.
- Shirley, J. N., Rubber Age 22: 71, 1927.
- Shull, J. R., Radiology 27: 279, 1936.
- Sikl, H., Ztschr. f. Krebsforsch. 32: 609, 1931.
- Sikl, H., Acta Unio internat. contra cancerum 6: 1366, 1950.
- Silson, J. E., Am. J. Public Health, New York 40: 943, 1950.
- Silson, J. E., Monthly Review. New York State Dept. of Labor 29: 41, 1950.
- Silson, J. E., Monthly Review. New York State Dept. of Labor 30: 5, 11, 1951.
- Silson, J. E., Benjamin, L. P. and Wilson, S. C., Monthly Review. New York State Dept. of Labor 28: 13, 1949.
- Siltzbach, L. E., Am. Rev. Tuberc. 55: 170, 1947.
- Silverman, S. B. and Erickson, C. C., Arch. of Path. 50: 63, 1950.

- Simmross, E., Virchow's Arch. f. path. Anat. 285: 183, 1932.
- Simons, E. J., Primary Carcinoma of the Lung. Publisher, The Year Book, Inc., Chicago, Ill., 1937.
- Singer, J. J., Surg. 8: 910, 1940.
- Sissons, H. A., Acta Unio internat. contra cancerum 7: 171, 1950.
- Sissons, H. A., 5th Internat. Congr. Du Cancer, Paris, 1950, 50 pages.
- Skavlem, J. H. and Ritterhoff, R. J., Am. J. Path. 22: 493, 1946.
- Sladden, A. F., Lancet 2: 123, 1933.
- Smith, L. W., Compensation Med. 2: 3, 1949.
- Smith, M. E., Atomic Energy Comm., Unclassified Document 1213; Abstract, Arch. Ind. Hyg. & Occup. Med. 4: 610, 1951.
- Smith, M. I., J.A.M.A. 116: 562, 1941.
- Smith, M. I., Franke, K. W. and Westfall, B. B., Public Health Rep. 51: 1496, 1936.
- Smith, M. I., Stohlman, E. F. and Lillie, R. D., J. Pharmacol. & Exper. Therap. 60: 449, 1937.
- Smith, W. E., Arch. Ind. Hyg. & Occup. Med. 5: 209, 1952.
- Smith, W. E., Sunderland, D. A. and Sugiura, K., Arch. Ind. Hyg. & Occup. Med. 4: 299, 1951.
- Snegireff, L. S. and Lombard, O. M., Arch. Ind. Hyg. & Occup. Med. 4: 199, 1951.
- Southam, A. H., Rep. of the Internat. Conf. on Cancer, London, 1928, p. 280. Publisher, John Wright & Sons, Ltd., Bristol.
- Spain, D. N. and Parsonnet, V., Cancer 4: 277, 1951.
- Spamer, E., Ztschr. f. Laryngol. 10: 44, 1921.
- Spannegel, H., Zent. f. Arbeitsmed u. Arbeitsschutz 1: 15, 1951.
- Spedini, F. and Valdini, P. L., Radiol. med. 26: 1, 1939.
- Ssipowsky, P. W., Ztschr. f. Krebsforsch. 36: 67, 1932.
- Staghelin, R., Klin. Wchnschr. 4: 1853, 1925.
- Steiner, P. E., Arch. of Path. 37: 185, 1944.
- Steiner, P. E., Butt, E. M. and Edmondson, H. A., J. Nat. Cancer Inst. 11: 497, 1950.

- Stephens, G., Med. Press & Circular, London 187: 194, 216, 283, 1933.
- Sterner, J. H. and Eisenbud, M., Arch. Ind. Hyg. & Occup. Med. 4: 123, 1951.
- Stewart, M. J. and Faulds, J. S., J. Path. & Bact. 39: 233, 1934.
- Stocklasa, J., Deutsche med. Wehnschr. 59: 1199, 1933.
- Stocke, P., 4th Ann. Rep. Brit. Empire Cancer Campaign 13: 257, 1936.
- Stocks, P., General Register Office. Studies on Medical and Population Subject: No. 1. H. M. Stationery Office, London, 1947, 46 pages.
- Stocks, P., Lancet 1: 351, 1951.
- Stocks, P., Brit. J. of Cancer 6: 99, 1952.
- Stokinger, H. E., Steadman, L. T. and Root, R. E., Am. Ind. Hyg. Assn. Meetings Chicago, April 1950.
- Stokinger, H. E., Sprague, G. F., Hall, R. W. and others, Arch. Ind. Hyg. & Occ Med., Chicago 1: 379, 1950.
- Stoll, R., Bass, R. and Angrist, A. A., Arch. Int. Med. 68: 831, 1951.
- Straube, G., Samul. v. Vergiftungsf. 10: 33, 1939.
- Strieck & Böhme, cited by Horning.
- Strnad, F., Monatschr. f. Krebsbekampf. 8: 309, 1938.
- Sweany, H. C., Porsche, J. D. and Douglass, J. R., Arch. of Path. 22: 593, 1936.
- Symanski, Arch. f. Gewerbepath. u. Gewerbehyg. 9: 295, 1938-1939.
- Symmers, W. St. C., Am. J. of Path. 27: 493, 1951.
- Syrek, A., Ztschr. f. Krebsforsch. 36: 409, 1932.
- Tabershaw, I. R., Arch. Ind. Hyg. & Occup. Med. 3: 298, 1951.
- Teleky, L., Deutsche med. Wehnschr. 62: 1353, 1936.
- Teleky, L., Klin. Wehnschr. 16: 910, 1937.
- Teleky, L., Am. J. Cancer 30: 385, 1937.
- Teleky, L., J. Ind. Hyg. & Toxicol. 19: 73, 1937.
- Teleky, L., Wien. klin. Wehnschr. 50: 619, 1937.
- Teleky, R., Occupation and Health, Internat. Labour Office, Suppl., Geneva, 193 p. 1.

- Teleky, L., Acta Unio internat. contra cancrum 3: 253, 1938.
- Teleky, L. and Gilbert, Occupation and Health, Vol. 2, Internat. Labour Office, Geneva, 1934, p. 227.
- Teutschlaender, O., Schlusswort zur Diskussion zu den Vorträgen p. 1. Ztschr. f. Krebsforsch. 32: 689, 1930.
- Thiele, A., Martin Pansa, Sachsen ältester Gewerbearzt. Ztschr. f. Öffentl. Gesundheitspflege 6: 348, 1921.
- Thiele, A., cited by Risel, Die Schneeberger Lungenkrankheit, in Fabrikärzte der chemischen Industrie. Ärztliche Merkblätter über berufliche Erkrankungen, 3rd edition, J. Springer, Berlin, 1930.
- Thiele, A., Rostoski, O., Saupe, E. and Schmorl, G., Muench. med. Wchnschr. 71: 24, 1924.
- Thiry, U., Arch. des maladies professionnelles. 2: 645, 1939-1940.
- Thomas, M. D. and Collier, T. R., J. Ind. Hyg. & Toxicol. 27: 201, 1945.
- Thys, Rev. Belge sc. méd. 7: 640, 1935.
- Timmermans, F. D., Zentralbl. f. Gewerbehyg. 8: 280, 307, 1931.
- Tonges, E. and Kalbfleisch, H. K., Frankfurt. Ztschr. f. Path. 50: 100, 1937.
- Touraine, A. and Bour, H., Rev. méd. franç. 20: 285, 1939.
- Tschelnitz, H., Strahlentherapie. 53: 269, 1935.
- Turner, F. C., J. Nat. Cancer Inst. 2: 81, 1941.
- Turner, H. M. and Grace, H. G., J. Hyg. 38: 90, 1938.
- Twort, C. C. and Fulton, J. D., J. of Path. & Bact. 32: 149, 1929.
- Twort, C. C. and Twort, J. M., Ztschr. f. Krebsforsch. 32: 491, 1930.
- Twort, C. C. and Twort, J. M., J. Ind. Hyg. 13: 204, 1931.
- Twort, C. C. and Twort, J. M., Lancet 2: 1226, 1935.
- Twort, J. M. and Twort, C. C., Ztschr. f. Krebsforsch. 32: 491, 1930.
- Twort, J. M. and Twort, C. C., Am. J. Cancer 23: 52, 1935.
- Uhlig, M., Virchows Arch. f. path. Anat. 230: 76, 1921.
- Urone, P. F., Druschel, M. L. and Anders, H. K., Analytical Chem. 22: 472, 1950.
- U. S. Dept. of Labor, Industrial Health Series No. 5, 1939.
- Vaccaro, L., Ind. Med. 10: 246, 1941.

- Van Ordstrand, H. S., *Modern Med.* 20: 71, 1952.
- Van Ordstrand, H. S., Hughes, R., DeNardi, J. M. and Carmody, M. G., *J.A.M.A.* 129: 1084, 1945.
- Van Ordstrand, H. S., DeNardi, J. M. and Schneider, R. W., *Cleveland Clinic Quarterly* 18: 48, 1951.
- Versluys, J. J., *Brit. J. Cancer* 3: 161, 1949.
- Vesin, M. S., *Arch. Mal. Prof.* 9: 280, 1948.
- Vigliani, E. C., *Folia med.* 23: 451, 1937.
- Vigliani, E. C., *Proc. 9th Internat. Congr. on Ind. Med.*, London, 645, 1948.
- Vinson, P. P., *J.A.M.A.* 107: 258, 1936.
- Volkman, R., *Publications of the D.G.F.C.*, Vol. 3: 3, 1874.
- Vollmann, J., *Schweiz. Ztschr. f. allg. Path. u. Bakt.* 1-2: 440, 1938-1940.
- Von Haam, E., Titus, H. L., Shinowara, S. H. and Caplan, I. T., *Fed. Proc.* 11: 432, 1952.
- Von Pein, H., *Deutsche Arch. f. klin. Med.* 190: 429, 1943.
- Vorwald, A. J., Editor. *Pneumoconiosis, Beryllium, Bauxite Fumes, Compensation.* Leroy U. Gardner Memorial Volume, Sixth Saranac Symposium. Publisher, Paul B. Hoeber, Inc., 1950, 659 pages.
- Vorwald, A. J., *Arch. Ind. Hyg. & Occup. Med.* 3: 1, 1951.
- Vorwald, A. J. and Karr, J. W., *Am. J. Path.* 13: 654, 1937.
- Vorwald, A. J. and Karr, J. W., *J.A.M.A.* 110: 2086, 1938.
- Vorwald, A. J. and Karr, J. W., *Am. J. Path.* 14: 49, 1938.
- Vorwald, A. J., Durkan, T. M. and Pratt, P. C., *Arch. Ind. Hyg. & Occup. Med.* 3: 1, 1951.
- Voss, H., *Arch. f. Gewerbepath. u. Gewerbehyg.* 9: 453, 1939.
- Wacker, L. and Schmincke, A., *Muench. med. Wchnschr.* 58: 1607, 1681, 1911.
- Wackmann, J., *Med. d. Lavoro* 24: 189, 1933.
- Waller, R. E., 28th Ann. Rep. Brit. Empire Cancer Campaign, p. 99, 1950.
- Waller, R. E., *Brit. J. Cancer* 6: 8, 1952.
- Walzer, I., *Ariz. Med.* 9: 30, 1952.

- Warren, I. I., Cancer Morbidity Series 3, U.S.P.H.S., N.C.I., 52 pages, 1951.
- Warren, L. and Spencer, J., Am. J. Roentgenol. 43: 682, 1940.
- Warren, S., Occup. Med. 5: 249, 1948.
- Warren, S. and Gates, O., Arch. of Path. 30: 440, 1940.
- Warren, S. and Drake, W. L., Am. J. Path. 27: 573, 1951.
- Wassink, W. F., Nederl. tijdschr. v. geneesk. 92: 3732, 1948.
- Weber, F. A., Die Bergkrankheit der Erzbergleute in Schneeberg in Sachsen. Arb. a. d. Reichsgesundheitsamt, 1926.
- Weber, L. F., Arch. Dermat. & Syph. 35: 129, 1937.
- Wedler, H. W., Klinik der Lungenasbestose, Publisher, Georg Thieme, Leipzig, 1937.
- Wedler, H. W., Deutsche. med. Wchnschr. 69: 575, 1943.
- Wedroff, N., Arch. f. Gewerbepath. u. Gewerbehyg. 6: 179, 1935.
- Wegelin, C., Schweiz. med. Wchnschr. 72: 1054, 1942.
- Weigl, A., Helvet. med. acta. 7: 142, 1940.
- Weil, C. S., Smyth, H. F. and Nale, T. W., Arch. Ind. Hyg. & Occup. Med. 5: 535, 1952.
- Weller, C. V., Arch. of Path. 7: 478, 1929.
- Wells, H. S. and Cannon, P. R., Arch. of Path. 9: 869, 1930.
- Welz, A., Arch. f. Gewerbepath. u. Gewerbehyg. 11: 536, 1942.
- Widmann, B. P., Am. J. Roentgenol. 47: 24, 1942.
- Williams, C. R., Proc. 9th Internat. Congr. on Ind. Med., London, 633, 1948.
- Willis, H. S. and Brutsaert, P., Am. Rev. Tuberc. 17: 268, 1928.
- Wilson, S. A., Rhode Island Med. J. 31: 719, 1948.
- Wilson, S. A., Radiology 50: 770, 1948.
- Winston, J. R. and Walsh, E. N., J.A.M.A. 147: 1133, 1951.
- Woglom, W. H., Arch. of Path. 2: 533, 708, 1926.
- Wood, E. H., Jr., Radiology 40: 193, 1943.
- Woodhouse, D. I., Brit. Empire Cancer Campaign, Ann. Rep. 1951, p. 170, published 1952.

- Woodhouse, D. L. and Irwin, J. O., *J. of Hyg.* 47: 121, 1950.
- Woodruff, C. E. and Nahas, H. C., *Am. Rev. Tuberc.* 64: 620, 1951.
- Wright, W. H., et al., *Nat. Inst. of Health Bull.* No. 189, U.S.P.H.S., 1947.
- Wyatt, J. P., and Riddell, A. C. R., *Am. J. Path.* 25: 447, 1949.
- Wyers, H., *Brit. J. Ind. Med.* 3: 177, 1946.
- Wyers, H., *Postgrad. Med. J.* 25: 631, 1949.
- Wyers, H., cited by Smith, W. E., *Arch. Ind. Hyg. & Occup. Med.* 5: 242, 1952.
- Wynder, E. L., *Deutsche med. Wchnschr.* 76: 1498, 1951.
- Wynder, E. L., *New England J. Med.* 246: 492, 538, 573, 1952.
- Wynder, E. L., *Arch. Ind. Hyg. & Occup. Med.* 5: 218, 1952.
- Wynder, E. L. and Graham, E. A., 5th Internat. Congr. on Cancer, Paris, p. 166, 1950.
- Wynder, E. L. and Graham, E. A., *J.A.M.A.* 143: 329, 1950.
- Wynder, E. L. and Graham, E. A., *Arch. Ind. Hyg. & Occup. Med.* 4: 221, 1951.
- Wyss, V., *Rass. Med. Indust., Turin* 19: 290, 1950.
- Young, A. M., Applebaum, H. S. and Wasserman, P. B., *J.A.M.A.* 112: 2406, 1939.
- Young, J. W., *Arch. Dermat. & Syph.* 49: 309, 1944.
- Young, M. and Russell, W. T., H. M. Stationery Office, 1926, London, Special Rep. Series No. 99, p. 20.
- Zanetti, E., *La Medicina del Lavoro* 31: 217, 1940.
- Zenker, F. A., *Tagebl. d. 40. Versamml. d. Natuforsch. and Ärzte*, p. 271, 1865.
- Ziel, R., *Med. Klin.* 26: 623, 1930.
- Ziel, R., *Med. Klin.* 31: 1535, 1935.

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Occupational and Environmental Pulmonary Cancer

With Special Reference to Pneumoconiosis

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National Institutes of Health

United States Public Health Service

Department of

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Bethesda, Maryland

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"Do you realize that within the lifetime of men now living, within a hundred years or one-hundred and thirty years at most, all the external conditions under which man lives his life on this earth have been more completely revolutionized than during all the ages of recorded history which preceded."

R. A. Millikan.

Occupational and Environmental Pulmonary Cancer
With Special Reference to Pneumoconiosis

W. C. Hueper

1. Increase in Lung Cancer Frequency.

It is generally recognized that exposure to certain exogenous physical and chemical agents plays an important role in the causation of cancers of the skin. Considering the fact that the respiratory tract may be looked upon as an inverted part of the outer covering of the body and that many of the known or suspected cutaneous carcinogens also are inhaled, it is not surprising that observations made mainly during recent decades have brought to light a rapidly increasing number of environmental and occupational agents which are involved in the development of cancers of the nasal cavity, paranasal sinuses, larynx, and lung. The relative importance of these discoveries is greatly enhanced because of the consistent and spectacular increase in the frequency of cancer of the lung noted during the last 50 years in almost all industrialized countries (Hueper). This rise in the number of deaths from pulmonary cancers has reached in several regions such proportions, that cancer of the lung has replaced cancer of the stomach as the most frequent cause of death from cancer among males (Steiner, Butt and Edmondson; Halpert).

It has lately become a convenient expedient for some investigators to explain away unpleasant things in vital statistics, such as the rising death rate from bronchogenic cancer (Stocks) by changing fashions in certification of causes of deaths, increased awareness of the medical profession of lung cancer, progressive aging of the population and similar half and part truths. However, to the critical observer there exists little if any doubt that an appreciable part of this development is real. The following graphs (fig. 1 and fig. 2) and table (table 1) giving statistical data from this country and abroad support this statement.

Fig. 1. Death Rates for Lung and Larynx Cancer in the United States, 1925 - 1948.

Fig. 2. Lung Cancer Death Rates for England and Wales, 1900 - 1947.

These observations made on official death certificate data are in agreement with the evidence obtained by investigators using autopsy material, Steiner, Butt and Edmondson found at the Los Angeles General Hospital that carcinoma of the lung was noted during the period 1923 - 1927 in 0.6 percent of all autopsies and constituted 4.3 percent of all tumors, while in 1943 to 1946 corresponding figures were 2.3 percent and 11.3 percent. Similarly, Beeler and Iray observed at the Lotterman General Hospital in San Francisco bronchogenic carcinomas in 1.2 percent of all autopsies during the decade 1920 - 1929, whereas they occurred in 2.7 percent in the period 1940 - 1948.

Corresponding observations were recorded by Ochsner and DeBakey; Fosahn; Gowan; Metropolitan Life Insurance Company, and other American investigators (Hueper; Steiner; Bruby and Sweany). The statistical data of Dorn and Potter show that this trend has been in existence since 1914 and has shown a tendency in late years to become less pronounced (table 1).

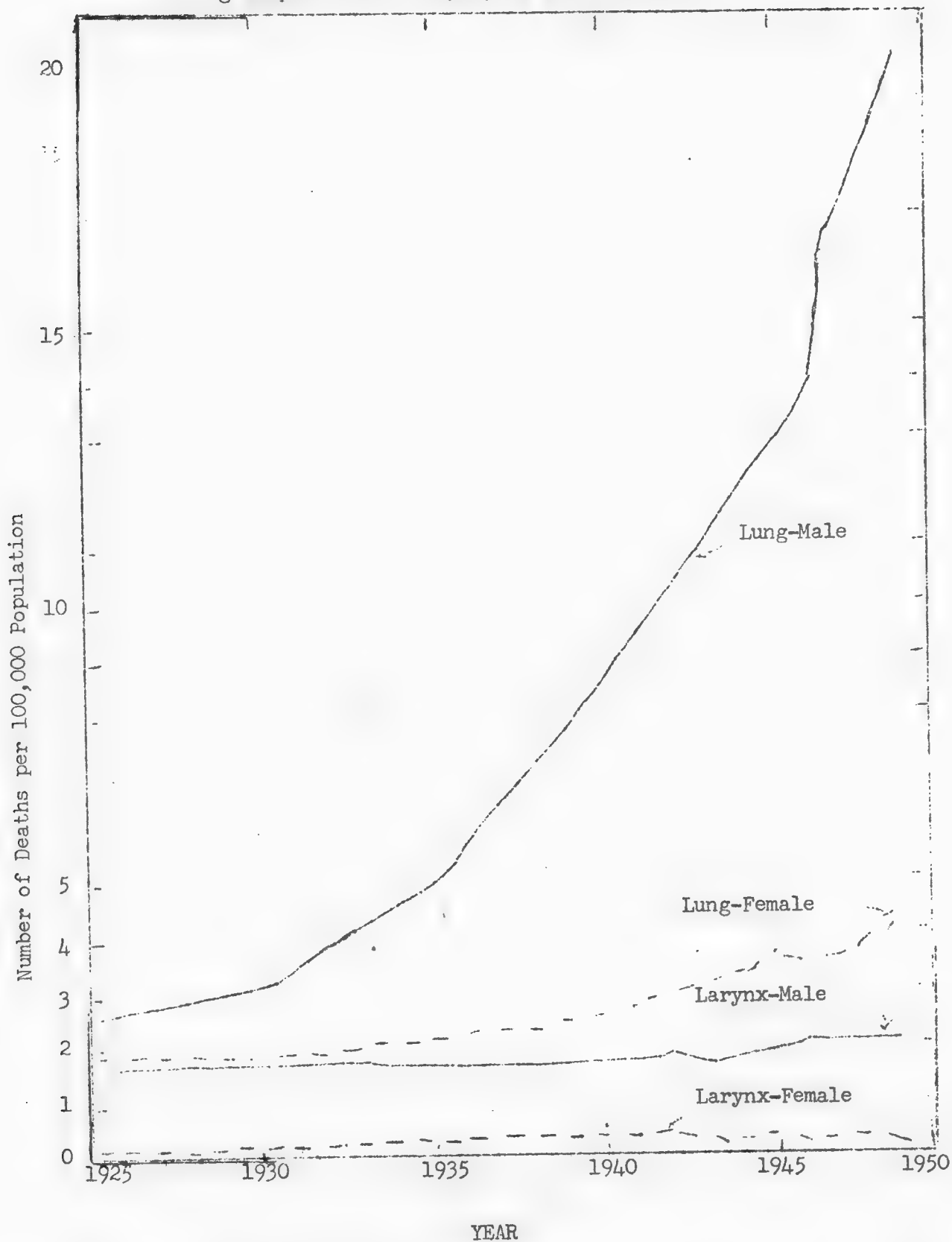
Schinz, Rosin and Sonti reported from Zurich, Switzerland, that the male mortality due to carcinoma of the lung between 1936 and 1944 was 9.8 per 100,000

Table 1.

Annual Age Adjusted Increase of Frequency of Lung Cancer Mortality

| <u>Dorn</u> | <u>1914-1930</u> | <u>1931-1940</u> | <u>Potter</u> | <u>1933-1944</u> |
|-------------|------------------|------------------|---------------|------------------|
| Males | 10.5% | 8.5% | | 5.8% |
| Females | 8.0% | 2.5% | | 2.0% |

Trend in Mortality From Respiratory Cancer,
Lung compared with Larynx, by Sex, 1925-1928



whereas it was 2.7 per 100,000 from 1896 to 1905. Brandt noted that in Riga, Latvia, the lung cancer incidence rose from 1.5 percent of all cancers found at autopsy in 1901 - 1905 to 10.5 percent in 1921 - 1925. Simross' analysis of the post-mortem material of Goettingen, Germany, yielded similar information, since cancer of the lung constituted 2.6 percent of all cancer in 1906 to 1912, while they were 9.8 percent in 1927 - 1931.

According to a report of von Glinski in 1939, 6.82 percent of all cancers seen at autopsy in Stettin, Germany, involved the lungs. Clemmesen evaluating official Danish mortality figures noted an apparent rise in lung cancer among males, whose attack rate increased from 5 per 100,000 in 1931 in Copenhagen to about 25 per 100,000 living in 1945, while the corresponding figures for females amounted to 4 and 7 per 100,000 respectively. The observations of Husted and Billman on Danish post-mortem material confirmed this trend. The investigations of Henschen on the autopsy material of Stockholm hospitals (Sweden) also revealed a progressive increase of lung cancer during the period 1900 to 1946. During the period 1900 - 1909, 1.6 percent of all cancers affected the lungs, during 1910 to 1919 the incidence figure stood at 2.1 percent and had risen to 5.85 percent for the period of 1920 to 1929. In the autopsy material of the St. Erik's Hospital in Stockholm, 9.9 percent of all cancers observed during 1937 to 1946 were located in the lung. While Casole reported in 1927 that among 2,658 necropsies performed in Padua during 1914 and 1925 there were two cancers of the lung (0.07 percent) and among 11,968 autopsies made during 1910 - 1925 in Milan, there were 15 pulmonary cancers (0.13 percent), Fabris noted in 1938, 150 lung cancers among 10,000 necropsies seen in ten years in Venice (1.5 percent).

The statistical studies of Stocks have demonstrated that also in England and Wales there has occurred a phenomenal increase in the frequency of lung cancer during the last several decades (fig. 2). Cancer of the lung in

England and Wales had standardized death rates of 1.1 for males and 0.7 for females during 1901 - 1920 and 10.6 for males and 2.5 for females in 1936 - 1939, which are almost identical with those recorded by Schinz for Zurich. These observations are in a general way confirmed for Great Britain by the studies of Kennaway; Kennaway and Kennaway; Heady and Kennaway; and Cheoseman as well as by those of Bonser. This investigator noted that there was a noticeable increase in the incidence of intrathoracic cancers at post-mortem at the Leeds General Infirmary during 1928 to 1937, following a long period (1891 - 1927) of relatively steady incidence, for which (1894 - 1928) also Passey and Holmes, using post-mortem material of various hospitals, did not observe any significant changes. Heady and Kennaway pointed out that there occurred a ninefold increase of cancer of the lung in men in England and Wales between 1928 and 1947, according to data of death certificates.

The evidence based on death rates from lung cancer and on autopsy observations was confirmed by the results of recent morbidity studies on cancer of the respiratory tract for eight metropolitan centers in the United States (Dorn; Warren; Grodowitz; Cutler; Marcus). The surveys conducted first in 1937 and repeated in 1947, showed for all metropolitan centers a consistent and often considerable increase in the morbidity rates of both cancers of the lung and, to a lesser extent, of the larynx (table 2). The rise in larynx cancer morbidity in the urban areas in the United States demonstrated by the surveys between 1937 and 1947 appears to be definitely more pronounced than the increase of crude larynx cancer death rates per 100,000 in the United States (1930: males, 1.42; females, 9.22; 1937: males, 1.70; females, 0.25) (Jackson and Jackson). These observations on the trend of death rates of larynx cancer in the United States are in relatively close agreement with those made in England and Wales (Kennaway and Kennaway) which stood in 1932 at 4.4 per 100,000 males and at 1.1 for 100,000 females, while they were in

Table 2.

Incidence of Respiratory Cancer, 1937 and 1947
Morbidity Rates for Eight Metropolitan Centers, by Sex
per 100,000 population*

| Primary Site and City | Males | | | Females | | | Total | | |
|--------------------------|-------|------|---------------------|---------|------|---------------------|-------|------|---------------------|
| | 1937 | 1947 | Percent Increase | 1937 | 1947 | Percent Increase | 1937 | 1947 | Percent Increase |
| <u>Bronchus and Lung</u> | | | | | | | | | |
| Atlanta | 5.0 | 13.4 | 168 | 1.0 | 5.0 | 400 | 2.9 | 8.9 | 207 |
| New Orleans | 13.1 | 39.1 | 198 | 2.8 | 4.2 | 50 | 7.6 | 20.8 | 174 |
| Dallas | 5.9 | 29.0 | 392 | 0.5 | 6.4 | 1180 | 3.1 | 17.2 | 455 |
| Birmingham | 4.5 | 18.9 | 320 | 2.1 | 3.9 | 86 | 3.3 | 11.0 | 233 |
| Denver | 9.1 | 21.9 | 141 | 4.2 | 8.1 | 93 | 6.6 | 14.8 | 124 |
| San Francisco | 15.6 | 34.3 | 120 | 3.9 | 8.1 | 108 | 9.8 | 20.8 | 112 |
| Chicago | 13.3 | 29.5 | 122 | 4.3 | 7.0 | 63 | 8.8 | 18.0 | 105 |
| Pittsburgh | 9.7 | 26.1 | 169 | 4.9 | 5.5 | 12 | 7.3 | 15.6 | 114 |
| Detroit | 12.6 | 32.0 | 154 | 2.3 | 5.7 | 148 | 7.5 | 19.0 | 159 |
| <u>Larynx</u> | | | | | | | | | |
| Atlanta | 1.4 | 4.0 | 186 | 0.3 | 0.3 | | 0.9 | 2.0 | 122 |
| New Orleans | 11.3 | 14.9 | 32 | 0.4 | 1.0 | 150 | 5.6 | 7.6 | 36 |
| Dallas | 3.2 | 5.3 | 66 | 1.5 | 0.4 | 73 | 2.3 | 2.7 | 17 |
| Birmingham | 1.4 | 4.0 | 186 | 0.0 | 1.3 | | 0.7 | 2.6 | 271 |
| Denver | 2.0 | 4.1 | 105 | 0.0 | 0.0 | | 0.9 | 2.0 | 122 |
| San Francisco | 4.5 | 8.8 | 96 | 0.2 | 0.8 | 300 | 2.4 | 4.6 | 92 |
| Chicago | 6.7 | 7.0 | 4 | 0.4 | 0.6 | 50 | 3.5 | 3.7 | 6 |
| Pittsburgh | 4.4 | 8.0 | 82 | 0.4 | 0.8 | 100 | 2.4 | 4.4 | 83 |
| Detroit | 3.5 | 6.4 | 83 | 0.4 | 0.3 | -25 | 2.0 | 3.4 | 70 |

*Biometrics Section
National Cancer Institute

1949, 4.1 for males and 1.3 for females.

It is apparent from these data that whatever factors may be responsible for the remarkable rise in the frequency of lung cancer, they have not - or to a much lesser degree - affected the incidence of cancer of the larynx.

If all due and liberal allowance is made for the progressive aging of the population since 1900, for an increased awareness of the medical profession of pulmonary cancer, for improved diagnostic facilities, for better recording systems, and for decreased deaths from other diseases than cancer, there still remains a considerable balance of lung cancers which cannot plausibly be accounted for by these factors. Surely, improvement in the medical proficiency of the pathologic diagnosis of lung cancer can scarcely be advanced as a factor of importance, since the histologic diagnosis of lung cancer is in general not a difficult task and has been practiced with efficiency and competence for many decades in the well-organized pathological institutes of Europe, which serviced the large public hospitals with their sociologically and economically relatively stable patient populations. The numerous objections voiced by Machlin on statistical grounds against the claim of an increase of lung cancers, pertain to only a minor degree to those institutes. Since fundamental changes in the biologic composition and constitution of the population groups involved did not take place within a few decades during which the increase in lung cancer frequency occurred, there remain only alterations in environmental factors related to modern industrialization and living conditions which plausibly might have provided the main causation of this development (Boycott).

2. Geographical Distribution.

The concept that exogenous factors, entering the human environment some 75 to 50 years ago and acting in increasing intensity, are responsible for the recent increase in lung cancers is supported by additional epidemiologic

observations, related to the geographical distribution of pulmonary cancers. The data on the incidence of respiratory cancer, 1937 and 1947, in eight metropolitan centers (table 2) reveal striking differences in the lung cancer morbidity rates of different centers, the extremes being 39.1 per 100,000 population, males, 1947, in New Orleans and 13.4 per 100,000 in Atlanta.

The percentages of increase in frequency for these communities also were far from uniform. Since the most marked discrepancies on these two points occur among metropolitan areas (New Orleans and Atlanta) located in the same part of the country (Southern States), it is most unlikely that differences in the genetic-biologic composition of the populations or fundamental variations in the smoking habit between these populations can be responsible for them. It is, therefore, much more probable that occupational, industrial or other environmental factors related to living conditions account for these regional variations.

Similar variations appear if cancer mortality data for different areas of States are analyzed. When Colorado, for instance, is divided into three regions according to predominating types of occupational activities (eastern part with agriculture; central part with industry, western part with mining and ranching) (fig. 3), it appears that the highest lung cancer death rate exists in the central, industrialized portion (50 per 100,000 male deaths) while the lowest is found in the agricultural area (30 per 100,000), with the western mining regions occupying an intermediary position (32). Seelig and Benignus also demonstrated that lung cancer death rates for 1930 to 1934 were higher in the urban areas than in the rural ones of the forty-eight States.

Similar state-wide discrepancies in the distribution of pulmonary cancer appear, if the lung cancer death rates of the forty-eight States are compared (table 3).

SPECIFIC CANCER DEATHS IN MALES

MINING
RANCHING

INDUSTRY

AGRICULTURE

DEATH RATE
ALL CANCERS-147
PER 100,000
SKIN-15
RESPIRATORY-32

DEATH RATE
ALL CANCERS-200
PER 100,000
SKIN-15
RESPIRATORY-50

DEATH RATE
ALL CANCERS-150
PER 100,000
SKIN-13
RESPIRATORY-30

COLORADO - 1943 - 1949

- 6a -

Fig. 3.

Table 3.

Lung Cancer Death Rates in the 25 States of the United States
in 1946 and 1948
Crude Death
Rates per 100,000

| Industrialized States | | |
|-----------------------|------|------|
| State | 1946 | 1948 |
| Connecticut | 8.5 | 11.1 |
| Illinois | 8.1 | 8.2 |
| Maryland | 6.5 | 8.4 |
| Massachusetts | 10.4 | 10.2 |
| Michigan | 5.7 | 7.1 |
| New Hampshire | 7.4 | 10.1 |
| New Jersey | 9.7 | 9.7 |
| New York | 10.2 | 11.9 |
| Ohio | 6.0 | 7.9 |
| Pennsylvania | 6.7 | 8.4 |
| Rhode Island | 8.7 | 7.4 |

| States with Regional Industrialization | | |
|---|------|------|
| State | 1946 | 1948 |
| Florida | 6.8 | 7.4 |
| Louisiana | 6.5 | 8.5 |
| Missouri | 7.3 | 9.4 |
| Montana | 10.0 | 8.8 |
| Nebraska | 5.7 | 8.0 |

| Agricultural States | | |
|---------------------|------|------|
| State | 1946 | 1948 |
| Alabama | 4.0 | 5.1 |
| Arkansas | 3.6 | 5.4 |
| New Mexico | 2.6 | 3.0 |
| North Carolina | 3.1 | 4.0 |
| North Dakota | 5.6 | 4.1 |
| Oregon | 4.1 | 4.4 |
| South Carolina | 3.6 | 3.7 |
| Washington | 5.1 | 4.2 |
| Wyoming | 4.9 | 3.9 |

The death rates for the year 1946 were taken from "The American Cancer Society, Inc., 1949, Cancer Death Rates for each State in the United States by Site"; those for the year 1948 were produced by the National Office of Vital Statistics (Rigdon and Kirchoff).

The extremes in deviations of lung cancer death rates between different States were presented by New York with 11.9 per 100,000 deaths as the highest rate and Idaho with 2.9 in 1948. Although the differences are in part explainable by variations in medical care, recording, etc., they are too marked not to be in part at least, real and to represent actual trends.

It is remarkable, moreover, that rates above 7.1 are mainly met in industrial States (Massachusetts, Connecticut, Rhode Island, New York, New Jersey, Pennsylvania, Ohio, Illinois, Missouri and Michigan) or in States having known cancer-producing industries in some industrialized parts (Montana, Louisiana, Maryland), while the majority of the predominantly agricultural States having relatively low lung cancer rates.

Similarly, Springett reported that the mortality from lung cancer was five times higher in England and Wales than in Norway, while the mortality from larynx cancer in Norway was only one quarter that in England. Fischer, likewise, remarked on the considerable variations of the lung cancer frequency in different parts of Germany, where the highest incidence figures were reported from the densely populated and highly urbanized and industrialized districts of Saxony and the Ruhr Valley (Dissmann).

Stocks; Kennaway; Kennaway and Kennaway; and Fulton also noted in their more recent studies that there was a prevalence of cancers of the lung and larynx in urban areas over rural ones. In urban populations there was, moreover, a lack of influence of social class upon the liability to lung cancer. If the coefficient of number of persons producing one cancer death was set arbitrarily at one hundred for the administrative county of London during 1946 to 1949, it was 233 for rural districts (Kennaway). For cancer of the larynx the coefficients were 100 and 170, respectively. Stocks' statistical analyses of the lung and larynx cancer incidence for different age groups during the periods 1921 to 1930 and 1940 to 1944 revealed that cancer of the

respiratory organs was the certified cause of much higher death rates in urban than in rural areas. In fact, it was shown in 1936 that for cancer of the lung in males there was a steep downward gradient from London through large and small towns to rural areas. While the standardized mortality ratios for cancer of the larynx at ages 35 - 64 for social classes in England and Wales, 1930 - 1932 showed a definite increase toward the lowest social class, cancer of the lung revealed only small and insignificant correlations with the environmental and social indices as expressed by the five social classes used in the evaluation. From studies of the records of the Meteorological Office, however, it appeared that there existed a positive correlation between the lung cancer death rate and sunshine hours for 20 towns investigated. Stocks suggests that the only explanations of these results which seem adequate were that either smokiness of atmosphere is an important factor in itself in producing cancer of the lung, or sunshine is an important factor in preventing its incidence.

In a recent study of Mills on the distribution of respiratory tract cancers in relation to atmospheric pollution in Cincinnati, this author came to the conclusion that in general the cleaner suburbs of the city had low respiratory cancer rates, while the more industrialized, low lying districts had higher rates, and that the rates of respiratory cancer deaths per 10,000 males for a five-year period compared with the average carbon deposit per month in different parts of the city.

3. Atmospheric Pollution.

The topographical distribution pattern displayed by respiratory cancers clearly suggests the action of an environmental agent which is present or operative to a higher degree in urban and industrialized regions than in rural areas. Air pollution from effluents of domestic fireplaces, incinerators, industrial establishments, and carbon black plants, exhaust fumes from gasoline

and diesel engines and coal or oil-fired railroad locomotives, dust from asphalted, tarred and oiled roads and from abrasion of rubber tires (Sharrah), would perhaps best conform with this pattern. The possible causal significance of some or all of the mentioned sources of air pollution has been proposed previously by several investigators (Duguid; McCrae, Funk and Jackson; Klotz; Matz; Katz; Wegelin; Roffo; Seelig and Benignus; Kling, Samssonov and Heros; Oldofredi; Smith, Wm.; Lorentz). Others, however, disclaimed for various reasons, the existence of such connections (Husted and Billmann; Lehmann; Brandt; Fischer; Jaffe; Konrad and Franks; Kennaway and Kennaway; Stocks; Syrek) with lung cancer incidence in England; France; Germany, Poland, Latvia, Russia and Sweden.

The three main sources of potentially carcinogenic air pollution are represented by (a) the specific hydrocarbons which are contained in the combustion and distillation products of carbonaceous matter; (b) arsenicals released as fumes from metallurgical establishments (smelters), and coal-burning furnaces and power plants or as dust following their use as pesticides; (c) radioactive matter present as gases and fumes in the effluents from industrial and military radioactive operations, and radioactive reaction and decay products of atomic energy plants (Smith; Lowry).

a. Domestic soot, which may consist of up to 40 percent of tarry matter (Cohen and Ruston) and which is the chief atmospheric contaminant, contains, according to Goulden and Tipler, 3,4-benzpyrene (300 mg./kg. in a mixed sample) representing one of the carcinogenic agents present in coal tar and shale oil (Berenblum and Schoental). The same carcinogenic chemical has recently been demonstrated in carbon blacks which form a major constituent of automobile tires (Falk and Steiner). It has been estimated that a single automobile tire during its life, produces by friction with the surface upon which it travels, 750 billions of carbon black containing rubber particles

(Sharrah). This does not include the dust created from tarred, asphalted and oiled road surfaces on which the tire was worn. It has been established also that the exhaust of gasoline and diesel engines contains benzpyrene (Waller) and was demonstrated in automobile lubricating oil. It is noteworthy that whilst the particles of coal smoke are distributed over a large range of sizes, Waller found those in the exhaust of internal combustion engines concentrated in a limited range of small sizes. Dust particles of small size are known to undergo maximum retention in the lung.

Recent studies of Waller showed that samples of smoke drawn from the air at eight different towns in England contained benzpyrene. The concentration of benzpyrene rose sharply during the winter, and there was a tendency for the mean annual values to increase with the size of the town. The average benzpyrene concentrations during smog days increased fourfold (from 7.2 mg. per 100 m³ to 32.8 mg.). A large part seems to come, in the opinion of Waller, from domestic fires. Since it has been detected also in the exhaust of combustion engines, some benzpyrene in the atmosphere must come from this source.

While the human evidence concerning the existence of a causal relation between the inhalation of atmospheric carcinogens from soot, rubber tires and engine exhausts and cancer of the lung is suggestive (Schnurer), there exists adequate and valid proof of the carcinogenic properties of soot or carbon black or their benzolic extractives when applied to the skin of mice or inhaled into the lungs of experimental animals. Extracts of dust from the air of eight large industrialized American cities when injected subcutaneously into mice produced sarcomas at the site of introduction (Leiter and Shear; Leiter, Shimkin and Shear).

Mice exposed to the inhalation of soot obtained from a hospital flue stack developed an excessive number of pulmonary adenomas and carcinomas (Seelig and Benignus). Similar results were obtained by McDonald and Woodhouse as well as

Campbell, when mice repeatedly inhaled clouds of soot collected from an English city or swept from tarred English roads. Squamous cell cancer of the bronchi was obtained by Muller in six (6) out of 24 rats painted on the skin with tar over prolonged periods, while Pessano reported similar results in rats exposed to the inhalation of the exhaust of combustion products of petroleum.

b. Extensive pollution of the air with arsenical effluents from metal ore smelters was especially in past decades, a well-recognized fact giving rise to damage to crops and wild and domesticated animals (Hofmann; Prell; Nieberle). It was an unavoidable while usually circumscribed complication of large scale dusting and spraying operations with arsenical pesticides. The contamination of the air of cities with arsenical impurities from the combustion of coal doubtlessly is in general of a much lower order. Goulden, Kennaway and Urquhart recently determined the arsenic content of the air obtained from eight (8) English cities. It was found that there were in one cbm. of air, 0.055 micrograms of arsenious oxide. This pollution increased during November to January to 0.104 micrograms. These amounts when inhaled by man over a period of years are considerably below those introduced into the lungs from smoking cigarettes or entering the body when Fowler's solution is taken. Goulden et al. believe, therefore, that the arsenic contained in the air of large cities may have at best a summation effect.

The human epidemiologic evidence on pulmonary cancer caused by an environmental arsenical air pollution is practically non-existent, unless the observations made during recent years in several counties in Montana having copper ore smelters provide what might be considered suggestive evidence (table 4).

c. Contamination of the atmosphere with radioactive material on a regional level near atomic energy plants or occurring at times on a wider scale as the result of massive discharges of radioactive matter following atomic

Table 4.

Lung Cancer Mortality in Several Counties
of Montana, 1947 - 1948

| County and Total Population 1940 | Major Industry | Number Lung Cancers | | | Total Cancer Deaths | % Lung Cancer | | Annual Lung Cancer Death Rate/100,000 | |
|---|------------------------------|------------------------|--------|-------|------------------------|------------------|--------|---|--------|
| | | Male | Female | Total | | Male | Female | Male | Female |
| Deer Lodge 13,627 | Copper Smelting | 21 | 0 | 21 | 98 | 30.8 | 0.0 | 145.7 | |
| Silver Bow 53,207 | Copper Mining | 27 | 2 | 29 | 259 | 22.6 | 1.5 | 48.6 | 3.9 |
| Cascade 41,999 | Copper Mining Smelting | 20 | 5 | 25 | 299 | 12.7 | 3.5 | 46.3 | 12.3 |
| Gallatin 18,269 | Agriculture | 1 | 0 | 1 | 81 | 3.0 | 0.0 | 5.2 | |

The estimated crude death rate for lung cancer among white males in the entire United States in 1947 is 10.9 per 10,000 population.

bomb explosions represents a special problem which still needs a great deal of critical investigation for properly assessing the possible creation of cancer hazards to the lung and other organs from prolonged exposures and cumulative effects. The ordinary radioactivity of the air, on the other hand, stems from the gaseous decay products of the small amounts of uranium and thorium distributed throughout the earth's crust. Dawson recently determined the degree of radioactivity of the suspended matter in the air of urban and rural areas in England. The amounts found were small (1/10th to 1/3000th of the lowest amounts considered harmful to man). It appears, therefore, that these common radioactive contaminants of the air do not play any significant role in the production of the ordinary type of human lung cancer. There is thus a certain amount of evidence suggesting the existence of a causal relation between some atmospheric pollutants with the relative distribution of respiratory cancer upon large groups of the general population.

4. Occupational and Professional Distribution, Dusty Trades and Tobacco

Smoking Habit.

Numerous attempts have been made also to establish evidence indicating an excessive lung cancer liability for special and restricted population groups related to occupations, trades, professions and habits.

a. Occupational Aspects. A considerable number of investigators recorded failure in attempts to find specific occupational relations to lung cancer incidence (Brockbank; Hollingsworth; Simmross; Haintz; Husted and Billmann; Jaffe; Rice; Rogers; Shennan; Bonser; and others). Others concluded that workers exposed to road dust or nonspecific industrial dusts and fumes displayed an excessive frequency of lung cancer and/or cancer of the larynx (Campbell; Duguid; Ferenczy and Matolcsy; Kennaway and Kennaway; Perrone and Levinson; Rosedale and McKay; Schachter; Singer; Aske-Upmark; Singer; Hampeln; Harvey; Seyfarth; Hudson; Rostoski; Saupe and Schmorl; Schmorl). Silica dust

in particular was mentioned by Weigl; Schmorl and Singer. The basic concept underlying these claims is apparently the still rather wide-spread, although erroneous belief, that cancer may develop on the basis of any nonspecific chronic irritation. Since occupational and experimental cancer research has rather definitely established the fact that carcinogenic properties are possessed by only specific agents of which only a part have also an appreciable irritative action, the above-mentioned assertions as to an alleged carcinogenic action of all and any kinds of dust seem to be conclusions drawn from pre-conceived ideas; and, therefore, are of relatively little scientific value. Street dust does not possess any carcinogenic properties in the opinion of Kikuth; Berblinger, Schmidtman and Probst. The latter cited in support the relative infrequency of lung cancer among policemen, trolley-men, teamsters, chauffeurs, street workers, and street vendors.

Of distinctly greater significance, on the other hand, are the observations made in regard to the increased or decreased frequency of lung cancer among members of certain occupational groups, especially as these data reveal a definite degree of uniformity with which certain worker groups are cited for their excessive liability although the data are coming from different investigators and obtained from different material. The information on this subject is summarized in table 5.

In addition to these occupational groups for which a certain amount of agreement exists, there were mentioned by some investigators, other groups which do not have general support. Versluys noted diamond cutters; Dublin and Graham, engravers; Wynder and Graham as well as BECC 1952, cabinet makers and carpenters; Wynder and Graham, smelter workers; and Versluys, butchers and barmen. A low frequency of lung cancer is recorded for agricultural workers (Kennaway and Kennaway; Versluys) and coal miners (Kennaway and Kennaway; Mason; Versluys; Feil; Schulte; Schulz; Allen).

Table 5.

Occupational Groups with an Excessive Lung Cancer Incidence

| Occupational Group | Investigator |
|--|---|
| Metal workers, welders, metal grinders and polishers, wire makers, tool and die makers, foundry workers, metal moulders, lathe workers, etc. | Borst; Kennaway and Kennaway; Turner and Grace; Mueller; Dublin and Vane; Wynder and Graham; McLaughlin |
| Cigar manufacturers and tobacconists | Seyfarth; Borst; Kennaway and Kennaway; Enger; Versluys; Brinkmann |
| Engineers; mechanics; machinists, plumers, etc. | BECC* 1944 and 1952; Gillespie; Turner and Grace; Mueller; Wynder and Graham |
| Painters, decorators | BECC 1944; Mueller; Dublin and Vane; Fulton. Wynder and Graham |
| Tar workers, road workers, asphalters, paviours, stokers, patent fuel workers, furnace men, foundry laborers, rollers, etc. | Kennaway and Kennaway; Fulton; BECC 1952 Registrar-General (1938), McLaughlin |

*Report of British Empire Cancer Campaign.

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*Report of British Empire Cancer Campaign.

Table 6

Lung Cancer Death Rate per 1,000 Deaths of All Causes for Seven (7)

Industrial Groups in Ohio, 1947 Among 5,309 Male Cancer Deaths

| Industry | % Respiratory Cancer |
|----------------------|----------------------|
| Iron and Steel | 2.18 |
| Transportation | 2.91 |
| Agriculture | 0.82 |
| Rubber and Plastics | 2.34 |
| Stone, Clay, Glass | 0.66 |
| Non-Ferrous Metal | 3.22 |
| Mining and Quarrying | 1.53 |
| Total | 1.76 |

Table 7

Lung Cancer Frequency Among Operating and Non-Operating Railroad Workers

| Railroad | Period | Total No. Lung Cancers | Lung Cancer Operating RR Workers | | Lung Cancers Non-Operating RR Workers | | Lung Cancers Unde- termined RR Workers | |
|----------|-----------|---------------------------|--|----|---|----|---|----|
| | | | No. | % | No. | % | No. | % |
| A | 1940-1950 | 29 | 24 | 83 | 5 | 17 | | |
| B | 1939-1949 | 104 | 59 | 56 | 15 | 15 | 30 | 29 |

The employment ratio of operating railroad workers to non-operating railroad workers of one of the two companies was 1:4. From this ratio it appears that about 75 percent of the lung cancers listed for railroad employees of these two companies occurred among the operating group which represents only 25 percent of the total number of employees. Operating railroad workers included engineers, firemen, conductors, men in the roundhouses and switchmen.

It may be mentioned finally that Bourne and Rushin demonstrated the presence of not inconsiderable amounts of chromium (chromite ore and chromates) in the atmospheric air in the immediate environment of a large chromate plant and that Davis recently reported that the use of soluble chromates as corrosion and rust inhibitors in automobiles had led to a yellow discoloration of the snow in Akron, Ohio. In view of the established high lung cancer liability of chromate workers, serious attention must be given during the coming years to possible carcinogenic effects resulting from such atmospheric pollution of the air with chromium containing carcinogens (Hueper). Such observations should not be lightly passed off with the unfounded assurance that it is inconceivable that any hazardous concentrations of chromium or arsenic or other carcinogenic agents might enter the air in the environs of plants (Foulger). Thorough and critical surveys must establish the harmlessness of such contaminations as definite facts before they can be dismissed as potential public health hazards. The occurrence of a not inconsiderable number of "neighborhood cases" of berylliosis caused by plant contamination of the environmental air and associated with at least a potential lung and bone cancer hazard for the victims should represent a very impressive warning against undue complacency.

In view of the demonstration of atmospheric carcinogens (Editorial, Lancet) the serious problem of the possible important role of industrial air pollution in the rise of lung cancers should not be characterized as a subject for "loose and injudicious statements". It is poor scientific judgment if persons

having serious and competent concern with a public health hazard of rapidly growing importance are suspected of "misusing statistics for propaganda purposes" (Lanza).

It is obvious from the evidence available that the incidence of lung cancer prior to 1900, in relation to the frequency of cancer in general, stood at approximately one percent of all cancer. After this period there was at first a gradual increase in the frequency of these neoplasms, followed in many localities studied by a sudden and much more rapid increase, the gradient becoming constantly steeper during the last three decades of this century. One of the characteristic features of this phenomenon is that the development did not start in different cities, countries and continents at the same time but that there were marked differences in the time of onset not only between different countries, but also between different regions and cities of the same country. The development, moreover, was not uniform in degree and intensity in different localities, but evidently affected urban, industrialized areas to a higher extent than rural ones and certain occupational groups more than others (Hueper; Steiner). If the action of environmental carcinogens should mainly account for the striking increase of lung cancer frequency and for its irregular course in different regions and conditions, industrial and industrially related carcinogens would well fit this pattern, since the growth of industrial establishments and the use of their products in the economic life of different countries and communities have greatly lacked uniformity in time, type and extent (Hueper).

b. Tobacco Smoking Habit. Rather far-reaching, if not extravagant, claims recently have been advanced as to the important, if not predominant role which cigarette smoking is alleged to have played in the production of lung cancer and its progressive rise in frequency during the past 50 years. A critical and sober analysis of the evidence offered in support of these

assertions is in order not only for reasons of scientific accuracy but also for medicolegal reasons and especially for determining the direction of future epidemiologic research and of control activities in the field of lung cancer.

A few years after there developed a growing appreciation of the rapid increase of cancer of the lung, smoking of tobacco, particularly, cigarettes, was suspected by some investigators as one of the causes, or the main cause of this phenomenon (Perret; Adler; Wassink, Arkin and Wagner; McCord; Bogen and Loomis; Grace; Thys; Syrek; McNally; Strnad; Joannovic; Berblinger; Hochstaetter; Schoenherr), although such connections were denied by Staehelin and Hintze.

Lickint in 1930, however, was the first to make definite claims in this respect, for which he sought support in the observations of Seyfarth and of Young, Russell, Brownlee and Collis concerning the excessive frequency of lung cancer among restaurant owners and waiters professionally exposed to tobacco smoke. These assertions were repeated by Hoffman; Ferrari; Brockbank; Roffo; Müller; Weigl; and Lehmann, as to the lung, and by Roffo; Herrmann; and Jackson and Jackson as to the larynx. Müller did the first statistical study on the relation of tobacco smoking to lung cancer by comparing the relative intensity of the smoking habit (cigarettes, cigars, pipe) among the members of a series of 86 lung cancer patients with the intensity distribution among a normal control group (table 8).

According to the occupational data given, there were in the cancer series 19 male individuals occupationally exposed to metal dusts and fumes, lubricating oil mist and soot; 12 exposed to soot and automobile exhaust; 11 exposed to ingredients of paints; one (1) exposed to chromates; while of the 10 female cancer cases, three (3) had worked in an ammunition plant and one (1) in a cigarette factory. A possibly significant occupational exposure history thus existed in 43 of the 76 male cases and in perhaps four (4) of the 10 female cases.

Table 8

Degree of Tobacco Consumption Among 86 Lung Cancer Cases and 86 Normal Controls

| Degree of Tobacco Consumption | Highly Excessive | | Very Heavy | | Heavy | | Moderate | | Non-Smokers | |
|---------------------------------------|---------------------|------|---------------------|------|---------------------|------|---------------------|------|---------------------|------|
| | <u>No. of Cases</u> | | <u>No. of Cases</u> | | <u>No. of Cases</u> | | <u>No. of Cases</u> | | <u>No. of Cases</u> | |
| % of Degrees Among Lung Cancer Series | 29 | (25) | 21 | (18) | 15 | (13) | 31 | (27) | 4 | (3) |
| % of Degrees Among Normal Controls | 5 | (4) | 6 | (5) | 25 | (22) | 48 | (41) | 16 | (14) |

An analysis of the tobacco smoking history of 93 lung cancer patients of Schairer and Schöniger revealed similar statistical correlations, since 29 were highly excessive smokers; 19 very heavy ones; 31 heavy ones; and 11 moderate ones, while three (3) were non-smokers.

While these discussions of a possible causal relation between cigarette smoking and lung cancer first aroused little attention beyond the narrow circle of research workers, the problem started to attract wide attention from the medical profession, public press, industry, and laity after the publication of the papers of Schrek, Baker, Ballard and Dolgoff and of Wynder and Graham in 1950. There followed in rapid succession a number of statistical investigations of this problem from this country and abroad (Levin, Goldstein and Gerhard; Breslow; Ochsner, DeCamp and DeBailey; Graham; Wynder; Mills and Porter; Dungall; Doll and Hill; Daff and Kennaway; Daff, Doll and Kennaway; Gsell). From the results of these studies the following conclusions were drawn by the different investigators:

Wynder and Graham: Excessive and prolonged use of tobacco, especially cigarettes, seems to be an important factor in the induction of bronchiogenic carcinoma. Among 605 men with bronchiogenic carcinoma, other than adenocarcinoma, 96.5 percent were moderately heavy to chain smokers for many years, compared with 73.7 percent among the general male hospital population without cancer.

Schrek et al.: The correlation between smoking and cancer is probably not due to fortuitous or secondary factors. It seems plausible, therefore, to formulate the hypothesis that there is a direct relationship between cigarette smoking and cancer of the respiratory tract and that cigarette smoking may be a carcinogenic agent. This relatively low percentage of deaths by cancer of the respiratory tract compared to the high percentage of smokers indicates that smoking is, at most, only a weak carcinogenic agent.

Ochsner, DeCamp and DeBakey: There is a distinct parallelism between the sale of cigarettes and the incidence of bronchogenic carcinoma. Because the carcinogenic effect of cigarette smoking does not become evident until after many years of smoking (approximately 20), it is frightening to speculate on the possible number of bronchogenic cancers that may develop as the result of the tremendous numbers of cigarettes consumed in the two decades from 1930 to 1950. If there is a causal relationship between cigarette smoking and bronchogenic carcinoma the deaths per 100,000 population from this cause may be expected to increase from 11.3 to 29.4 by 1970.

Levin, Goldstein and Gerhardt: These data support the conclusion that lung cancer occurs approximately 65 percent more frequently among males who have smoked cigarettes for 25 years or more than among males who have smoked cigars or pipes for a comparable period, or non-smokers. The data indicate also that pipe and cigar smokers have no higher incidence of lung cancer than non-smokers. The findings suggest, although they do not establish, a causal relation between cigarette and pipe smoking and, respectively, lung and lip cancer.

Mills and Porter: Among cancers of the respiratory tract from the larynx downward, an abnormally high percentage of cigarette smokers, as well as of pipe and/or cigar users, is found. This group of cancer victims exhibits significantly increased percentages in all forms of smoking.

Doll and Hill: Among the smokers a relatively high proportion of the patients with carcinoma of the lung fall in the heavier smoking categories. Smoking is a factor, and an important factor, in the production of carcinoma of the lung. The risk of developing carcinoma of the lung increases steadily as the amount smoked increases. If the risk among non-smokers is taken as unity and the resulting ratios in the three age groups in which a large number of patients were interviewed (ages 45 - 74) are averaged, the relative risks become

6, 19, 26, 49, and 65 when the number of cigarettes smoked a day are, 3, 10, 20, 35, and say 60 -- that is, the mid-points of each smoking group. Cigarette smoking was more closely related to carcinoma of the lung than pipe smoking. No distinct association was found with inhaling.

It appears from the speculations of Doll and Hill that among the population of Greater London over the age of 45, those who smoke 35 or more cigarettes a day, had a chance of developing cancer of the lung which was fifty (50) times greater than that of non-smokers of similar age. Assuming that these conclusions are essentially correct, it may then justly be argued that an effective control of cigarette smoking offers the means for a far-reaching prevention of cancer of the lung and, possibly, the larynx. The statistical data which form the basis of these conclusions are summarized in table 9. Brunner found among 127 lung cancer patients 75 percent heavy smokers and 9.5 percent non-smokers.

While some of the not inconsiderable differences in the relative percentages of smokers of various degrees are doubtlessly due to the use of different standards in the classification used, this explanation, however, does not hold for the proportion of non-smokers listed by the different investigators. The percentage range for non-smokers is from 1.3 to 14.6 percent for the various lung cancer groups and from 8.8 to 30.5 percent for the control groups. These discrepancies suggest the existence of differences in the basic composition of the human material evaluated. The validity of this concept also is supported by the fact that the various investigators noted rather widely varying proportions of adenocarcinomas in males and females in their respective series. The histologic type of pulmonary cancer is predominantly of the epidermoid variety among males, while a considerable proportion of these tumors among women are of the adenocarcinomatous kind (36.4% in females; 4.5% in males (Gsell); 52% in females; 18% in males (Proc. First Nat. Cancer Conf.); 13.7% in females; 6.7% in males (Mason); 52% in females; 0.6% in males (Wynder and Graham). It was noted also that a history of heavy smoking was less often

Table 9.

Statistical Correlations Between Tobacco Smoking and Lung Cancer
 Degree of Smoking Habit Among Lung Cancer Patients (Males)

| Authors | Highly Excessive | Very Heavy | Heavy | Moderate | Non-Smokers |
|------------------|---------------------|---------------|-------|----------|---|
| Schrek, et al. | 18.3 | 50.0 | | 12.2 | 14.6 Cigarettes only, balance: pipe & cigars. |
| Wynder & Graham | 20.3 | 30.9 | 35.2 | 12.4 | 1.3 |
| Doll & Hill | 5.0 | 21.0 | 30.3 | 38.6 | 5.1 |
| Breslow | 15.3 | 50.7 | 19.5 | 3.5 | 9.0 |
| Gsell | 30.0 | 37.0 | 21.0 | 10.5 | 2.0 |
| <u>Controls:</u> | | | | | |
| Wynder & Graham | 7.6 | 11.5 | 35.6 | 30.5 | 14.6 |
| Doll & Hill | 2.1 | 11.4 | 30.5 | 47.1 | 8.8 |
| Breslow | 3.5 | 34.8 | 18.8 | 11.1 | 30.5 |

- 21 -

elicited from patients with adenocarcinoma than in those with epidermoid carcinoma (Gsell; Wynder and Graham).

The apparent lack of uniformity in the human material analyzed by the different authors is further demonstrated by the appreciable differences in the sex distribution of lung cancers reported at different times, from different regions and by different investigators. The male to female sex ratio fluctuates between 2:1 to 20:1 (Hueper). It is noteworthy, however, that the uniformly observed prevalence of lung cancer among males has, in general, become in recent years even more pronounced than in former decades. This observation strongly militates against a predominant causal role of cigarette smoking in the production of lung cancer, because all previous experience in the field of occupational cancer indicates that given the same type of carcinogenic exposure for both sexes and at the same time an increasing equalization of the intensity of exposure, there occurs a narrowing of the gap in incidence rates of the two sexes and not a widening, which actually exists. This interpretation of the diverging, sex related frequency trends is not fundamentally affected by the statement that the interval between the start of tobacco smoking and the appearance of a lung cancer is between 20 and 40 years (Wynder and Graham; Ochsner, DeCamp and DeBakey; Schrek, Baker and Ballard). Even if women may not have indulged on a large scale in tobacco smoking some thirty years ago, there can be little doubt that the cigarette smoking habit has made during this period much greater strides among women than among men.

The purely statistical approach leading to the assumption of the existence of causal relations between two coincidental events and trends is thus in urgent need of supporting biologic evidence. It is for this reason that the negative statistical correlation between pulmonary cancer frequency and cigarette smoking recently reported by Dungal from Iceland has added relatively little to the basic issues. Dungal pointed out that there were among 1939 autopsies performed during 1939 - 1948 with 417 cancers of all sites only 12 pulmonary cancers (2.9% instead of 10 to 20% in the United States and 12%

in Switzerland), that tobacco, especially of cigarettes was not particularly popular in Iceland until 1939 and that tarring of the roads which had extensively been done since 1920, had not exerted any influence on lung cancer frequency.

Experimental Tobacco Cancer:

Attempts to produce cancers with tobacco tar in experimental animals began many years before even any relation between tobacco smoking and lung cancer was suspected - that is at a time when the claims of a causal relationship between cancer and tobacco smoking was still limited to cancers of the lip, tongue, mouth, and larynx (Martin, Friedell and Rosenthal).

a. Skin Applications of Tobacco Tar:- Macker and Schmincke as well as Helwig using tobacco tar extracts which they applied to the ears of rabbits and the skin of mice, respectively, produced only ulcers with epithelial proliferations, but not cancers. Similarly negative were experiments of Hoffmann, Schreus and Zurhelle who applied denicotinized tobacco tar for 80 days to the skin of mice; and by Cooper, Lamb, Sanders and Hirst who used the same technique for 23 months and observed a single skin cancer. Roffo and Chikamatsu reported the production of cancroids in the ears of a few rabbits after prolonged painting with tobacco tar. These claims were confirmed by experiments of Lu-Fu-hua but cancers of rabbits' ears were seen only when he used simultaneously intravenous cholesterol injections as well as painted the other ear with coal tar. These observations of Lu-Fu-hua on cholesterolized and coal tar treated rabbits were successfully repeated by Schürch and Winterstein who in turn failed to produce skin cancers in mice receiving skin application of tobacco tar and various tobacco tar fractions having different boiling points.

Sugiura subsequently succeeded in producing a solitary squamous cell carcinoma in a mouse painted with tobacco tar distilled at a temperature between 500°-900° C. Tobacco tar distilled between 100° - 500° C. proved to

be noncarcinogenic for mice when applied to the skin. Both distillates were administered in an oily mixture to the ears of rats and rabbits for 52 to 95 weeks without tumor formation. Flory repeated the application of tobacco tar distillates of different boiling points (130-350°C); 350-700°C) as well as of pipe tobacco tar to the ears of rabbits and obtained in a considerable proportion of the animals, papillomas and carcinomatoid tumors, but not carcinomas. Several squamous cell carcinomas were found in mice after the application of these tars.

b. Inhalation of Tobacco Tar Fumes:- The first attempts to produce cancer of the lung in experimental animals by the inhalation of tobacco smoke were made by Mertens using mice which were exposed in glass jars to tobacco smoke injected into these vessels. Among the first set of 125 mice, two (2) developed "lung cancer", but in both instances they most likely were of "spontaneous" origin. When this experiment was repeated no lung tumors were obtained. Likewise, negative were similar experiments of Lorenz et al. when mice inhaled tobacco smoke introduced into a closed container. The strain A mice exposed to tobacco fumes showed the same incidence of spontaneous lung tumors as control animals. In Campbell's experiments in which a similar technique of inhaling tobacco smoke was used, there followed a minor increase of lung tumors in the test series over that of the controls.

A more direct and drastic method for introducing tobacco tar into the lungs of experimental animals was chosen by Roffo who injected this material directly into the lungs of rats and obtained in one (1) rat, four (4) small squamous cell carcinomas.

A critical evaluation of the total experimental evidence permits the conclusion that tobacco tar obtained at various distillation ranges is of very weak if not doubtful carcinogenicity to the skin of mice and produces apparently only carcinomatoid tumors in the ears of rabbits, when applied over long periods

of time. The inhalation of tobacco smoke, released into the atmosphere, by mice failed to produce lung cancers. The unconfirmed positive claims of Roffo have been disregarded in reaching these conclusions.

c. Demonstration of Known Carcinogens in Tobacco Smoke and Tar.

Among the known carcinogenic chemicals which may occur in tobacco smoke or tar carcinogenic aromatic hydrocarbons and arsenicals have to be considered. The carcinogenic aromatic hydrocarbons might be formed during the combustion of the tobacco while arsenicals might occur in only those tobacco tars and fumes which are generated from tobacco containing arsenical insecticide residues.

Although Roffo asserted to have demonstrated 3,4-benzpyrene in tobacco tar by spectroscopic methods, this claim has remained unconfirmed by several very reliable investigators (Schürch and Winterstein; Cooper, Lamb, Sanders and Hurst; Waller).

While the failure to demonstrate 3,4-benzpyrene in tobacco tar does not exclude the possible presence of other carcinogenic chemicals in this material, it nevertheless is an observation which is noteworthy because 3,4-benzpyrene seems to be one of the common carcinogenic combustion products of carbonaceous matter of many kinds.

In view of these negative findings for carcinogenic hydrocarbons in tobacco tar some investigators recently have favored the concept that the alleged carcinogenic effect of tobacco smoke upon the respiratory tract depends at least in part upon the inhalation of arsenic present in the tobacco as an insecticide residue and volatilized during the smoking process (Doll and Hill; Goulden, Kennaway and Urquhart). In fact, rather appreciable amounts of arsenic can be demonstrated in tobacco and in tobacco smoke, especially of the American variety. Gross and Nelson found that the arsenic content of cigarette tobacco of five (5) brands ranged from 9.7 to 36.3 p.p.m., that of cigars from

8.3 to 48.4 p.p.m., and that of pipe tobacco from 26.0 to 50.0 p.p.m. Thomas and Collier noted that the range of the arsenic content of cigarette tobacco was from 35.4 to 114 p.p.m., while that of cigars was 13.2 to 29.5 and that of pipe tobacco 22.7 to 42.8 p.p.m. The reason for the marked variations in different samples and various types of tobacco is undetermined. However, different climatic conditions and methods of cultivation and processing of tobacco in various parts of the United States and the different use to which the various types of tobacco are subsequently put in the production of tobacco goods may have a decided influence in this respect.

In more recent studies of cigarette tobacco by Daff and Nelson, three (3) American brands gave an arsenious oxide content ranging from 25 to 47 micrograms; two (2) English brands had one ranging from 50 to 55 micrograms; eight (8) Turkish brands had one ranging from 0 to 4.1 micrograms; one (1) French brand ranged from 0.5 to 1.5 micrograms; and from a Rhodesian brand, the range was 1.8 to 4.1 micrograms. Popp found that Palatinate tobacco contained 5.1 p.p.m. of arsenic; Macedonian cigarette tobacco 0.7; Java tobacco 0.33; and Brazilian tobacco 4.6. Oliver reported that English pipe tobacco contained 32 p.p.m. of arsenic (as metal); cigarettes 68 p.p.m.; Jamaica cigars, 30 p.p.m.; and Havana cigars, 170 p.p.m.

The observations show that the concentration of arsenic in cigarettes, cigars and pipe tobacco is very variable depending upon the brand as well as upon the country of origin. The tobacco of American derivation, and smoked in the U.S.A., Canada, Norway and England has by far the highest arsenic content (24 to 106 micrograms As_2O_3 per gram of tobacco), while tobaccos grown in the eastern European countries and Turkey have, as a rule, a low arsenic content (0.0 to 4.3 micrograms As_2O_3 per gram of tobacco). This type of tobacco is used in cigarettes made in Austria, France, Poland and Bulgaria.

In adjudging the degree of arsenic hazard which may result from the smoking of arsenic containing tobacco, Daff and Kennaway, Thomas and Collier, and Gross

and Nelson ascertained that between 7.0 and 26 percent of the arsenic present in the tobacco is volatilized and may be inhaled during the smoking process. Remington, however, placed this portion as high as 50 percent. Daff and Kennaway expressed the degree of potential hazard by the following calculation:- "If a person smokes 50 cigarettes with a mean arsenic content of 50 micrograms and 15 percent of this escapes, he has volatilized 0.375 mg. As_2O_3 , which is the amount contained in 0.0375 cc. of Fowler's solution (official dose 0.125 to 0.5 cc.)."

In a recent, very illuminating study of the relation of cancer of the lung to tobacco as grown and smoked in different countries, Daff, Doll and Kennaway have unearthed important observations which strikingly demonstrate the complexity of the problem and show "that the arsenic content of tobacco has not provided any simple and exclusive explanation of the association between cigarette smoking and this form of cancer." Analyzing first the data provided by Saglam, Schwartz and Yenerman from Istanbul, Turkey, where for over five (5) decades tobacco was consumed almost wholly in the form of cigarettes and where there have been many heavy smokers among women, they found that there had been a considerable rise in lung cancer frequency during the past 50 years, according to clinical and anatomic-pathologic statistics (increase in clinical material, 12 times, in pathologic material, 4.1 times). However, the male: female ratio changed from 6:1 during 1935 to 1939 to 8:1 during 1949 - 1950, although cigarette smoking has been a habit indulged in by Turkish women for many decades. The tobacco consumption expressed in pounds per head increased from 1.21 in 1925 to 1.9 in 1949. Since the tobacco consumption stood in 1935 at only 1.55 lbs. per head, and in view of the long lag period in the development of lung cancer, it is most unlikely that the increase in lung cancer frequency in Istanbul has any relation either to tobacco consumption or to its arsenic content. This view is supported by the sex ratio which is, like in

many other countries, markedly and increasingly in favor of males, despite the long established smoking habit among Turkish women. Saglam also, therefore, rejected cigarette smoking as a factor in the incidence of bronchial carcinoma.

Another illustration of the apparent lack of significance of arsenic in tobacco in relation to the causation of lung cancer and its recent increase seems to be provided by the autopsy data from Ljubljana, Yugoslavia, provided by Kosir. During the period 1925 to 1939 lung cancer represented 7 percent of all males coming to autopsy with cancer of all sites, and 1.5 percent of all females of the same type, while during 1940 to 1949 these figures stood at 15 percent and 2 percent, respectively. Tobacco is smoked principally in cigarettes of oriental type with a low arsenic content. The increase in lung cancer frequency thus was disproportionately large for males.

The data provided to Daff, Doll and Kermaway on Switzerland by v. Meyenburg have been complemented by those recently given by Gsell.

According to Gsell, there has taken place in recent years a marked increase in the consumption of cigarettes (10-fold between 1924 and 1949) but the main tobacco product consumed in Switzerland had remained the "Stumpen", a medium-sized cigar without a tip. There were 48 Stumpen and cigar smokers and only 30 cigarette smokers among Gsell's 87 cases. The absolute number of lung cancer cases rose in males from 68 in 1905 - 1909 to 2058 in 1945 - 1949 (20.7 times), while the corresponding increase in female cancer deaths was from 59 cases in 1905 - 1909 to 421 cases in 1945 - 1949 (7.1 times). During the same period there occurred a shift of the male: female sex ratio from 2:1 in 1905 - 1909 to 4.9:1 in 1945 - 1949. The lung cancer death rate per million stood in 1931 at 66 for males and at 14 for females, while in 1947 it was 183 for males and 34 for females. The arsenic content of tobacco of Swiss cigarettes was of intermediate order (3.1 to 12.0 micrograms of As_2O_3 per gram). Since during the critical period of lung cancer increase for which Swiss tobacco consumption figures are available (1924 to 1935) the main use of tobacco was in the form of cigars, Stumpen and Toscani, one must assume that the majority of the lung

cancers observed during the years 1940 to 1949 cannot be attributed to the smoking of cigarettes, if smoking at all had any causal connection with their development. That the existence of such a relation is not likely is again attested by the divergent trends in the sex distribution of lung cancer, which according to past experience on this point from the field of occupational cancer would display a tendency toward equalization whenever members of both sexes are exposed to the same environmental carcinogenic agent. That this principle also applies to tobacco smoking is evident from the recent statistical studies of Doll and Hill who ascertained that the risk of developing cancer of the lung is the same in both men and women, apart from the influence of smoking.

The importance of this argument is strikingly illustrated by the information supplied to Daff, Doll and Kennaway by Kreyberg concerning the lung cancer incidence in Norway during the past 20 years. Apart from the fact that deaths from lung cancer were higher in urban than in rural districts of Norway, there occurred during the two decades a definite rise in pulmonary cancer frequency from about 20 in 1930 to approximately 157 in 1948. The rise was more marked in urban areas than in rural ones and affected males to a higher degree than females. While thus the Norwegian experience followed in this respect the usual pattern, it differed fundamentally from it in its sex distribution.

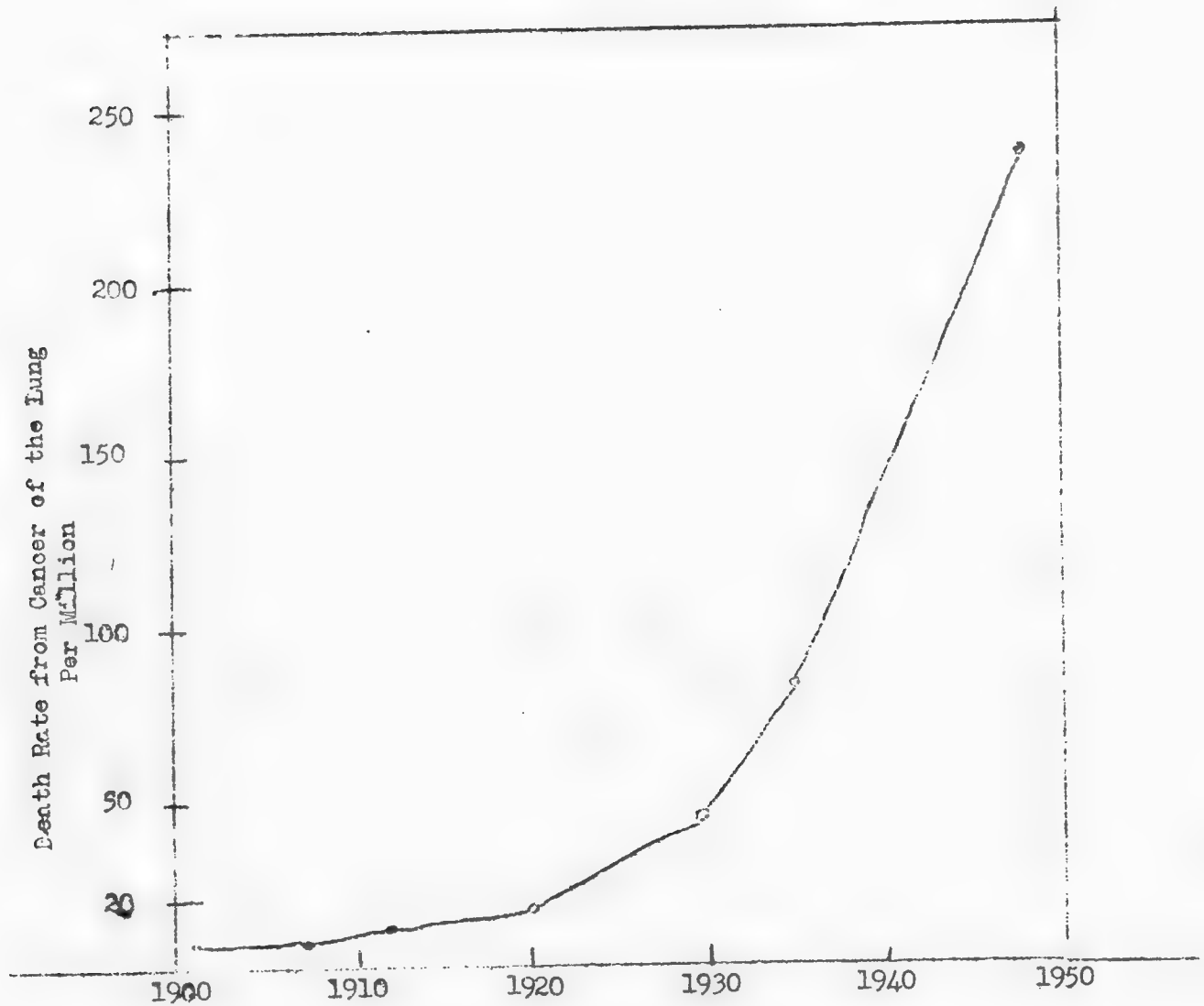
During the first six years of the survey period, lung cancers were apparently as frequent in females as in males and thereafter the rise of the rates in females fell somewhat below that of males. This observation permits only one interpretation when analyzed in the light of the existing facts, that the epidemiologic behaviour of lung cancer as to sex distribution was influenced in Norway by probably environmental factors which were not active at all or to the same degree in the other countries investigated. Since the population of Norway has smoked predominantly American made tobacco, this deviation from the common epidemiologic pattern does not support the view that tobacco smoking

in Norway or in any of the other countries was a deciding and important factor in determining lung cancer epidemiology during the past 50 years.

In the various arguments advanced purportedly favoring the cigarette smoking role in the causation and rise of lung cancers much is made of the existing parallelism between the increase of lung cancer frequency and the increased consumption of smoking tobacco, particularly cigarettes, during the last 25 to 50 years. In the graphic presentation of these two developments the relative events of annual rise of lung cancer rates and of tobacco consumption are invariably synchronized, while in fact these events have a distinct heterochronicity because of the long latent period of lung cancer which has been estimated for smokers to range between 20 and 40 years. The lung cancer cases observed in 1950, for instance, therefore have no causal connection with the tobacco consumption of the same year but more likely, if at all, with that recorded for 1920 to 1935. It is evident from this consideration that the so-called "parallelism" as presented by synchronized graph lines gives a definitely distorted expression of any possible hypothetical relation between lung cancer and tobacco consumption (figs. 4 and 5).

Distinctly disconcerting in this respect is also the obvious disagreement of different investigators as to the relative role which cigarette smoking, on the one hand, and the smoking of pipe tobacco and cigars, on the other hand, allegedly play in the causation and rise of lung cancer. While Levin et al. contended that only cigarette smoking but not pipe and cigar smoking reveals a positive statistical correlation, Mills et al. emphasized that all three forms of smoking are equally guilty, while Wynder and Graham; Gsell; and to some degree also, Doll and Hill, assess the individual smoking habit by including all types of smoking. While Doll and Hill contemplated the possibility that pipe smoking may be less lung cancer inducive than cigarette smoking because in their opinion pipe smokers smoke less tobacco than cigarette smokers,

Lung Cancer Death Rates
for England and Wales



The rates are based on three year averages for all years
except 1947
Doll, R. & Hill, A.B.: Brit. Med. J. 1: 739 (1950)

Fig.4.

Comparative Trends in Respiratory Cancer

Mortality 1924 - 1950

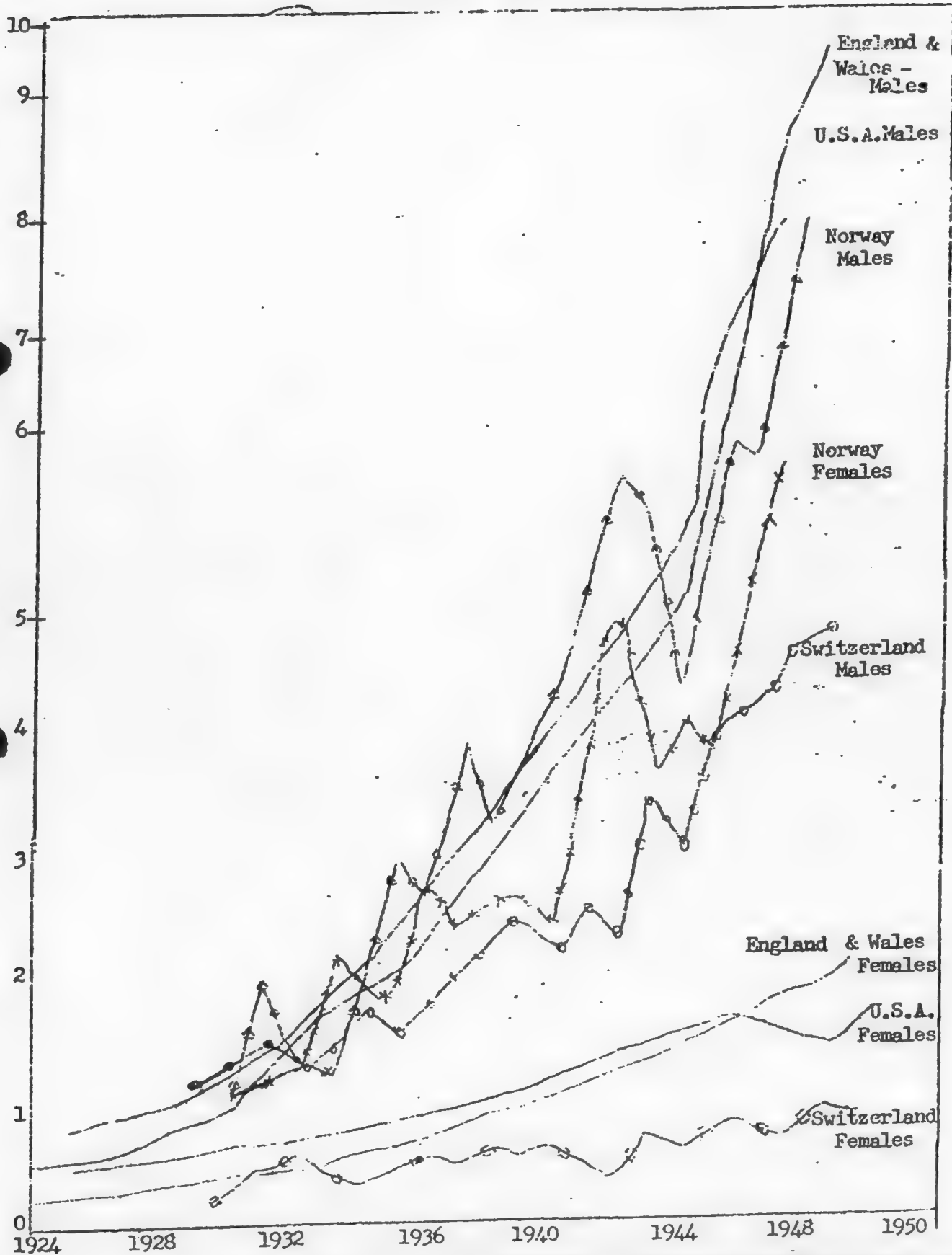
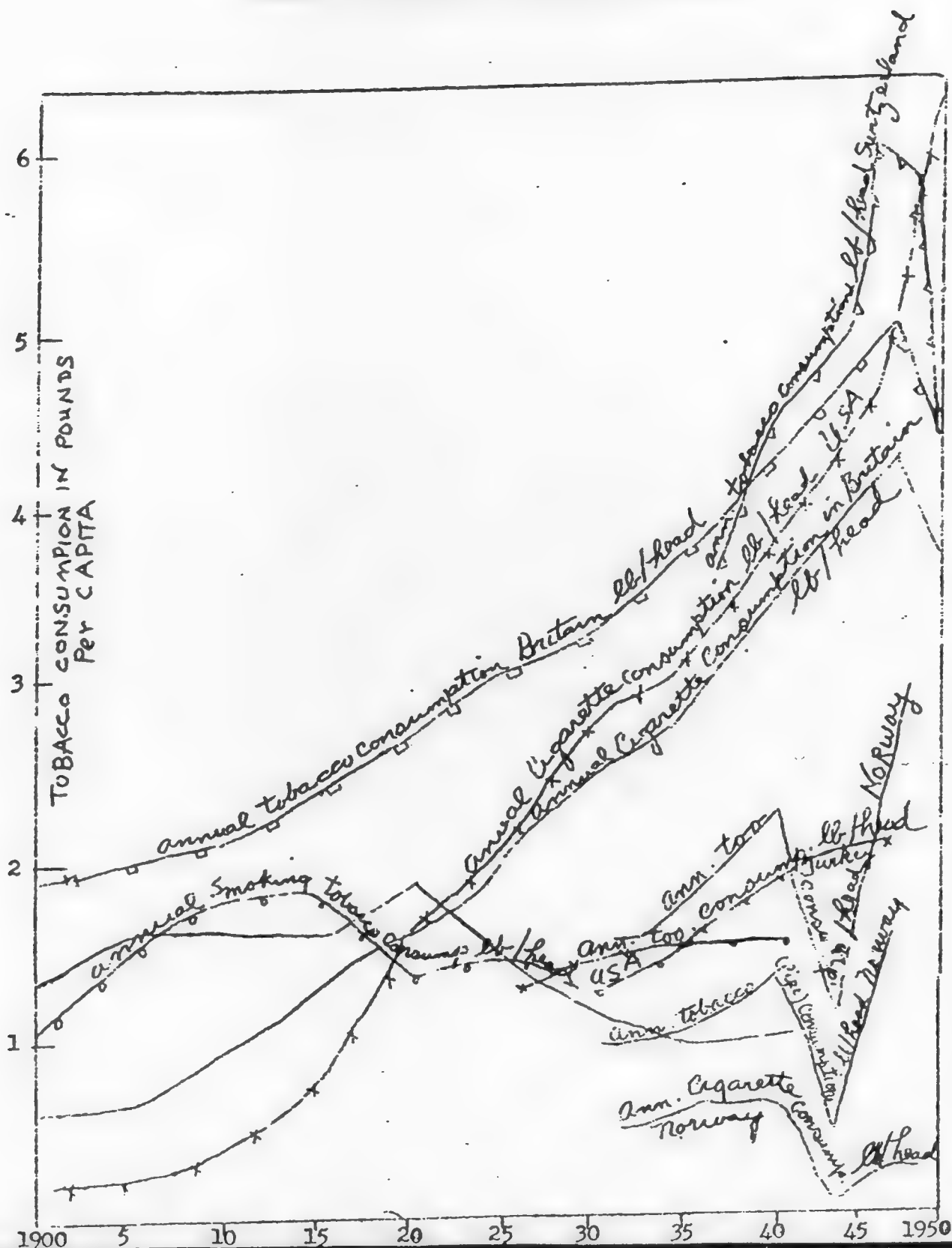


Fig. 5.

Tobacco Consumption per Capita in Pounds



it may be well to consider the fact that many cigarette smokers discard their cigarettes after a few puffs and that, therefore, the assessment of the degree of cigarette smoking may more easily become exaggerated, while that of the pipe and cigar smoker may become underestimated. Gage indeed stated that one study strongly indicated that the average cigarette smoker consumes less tobacco per day or year than a cigar smoker or chewer.

Of undoubted importance seems to be another statement of Doll and Hill in which they note that inhaling of cigarette smoke did not convey any increased lung cancer liability. This is an observation which cannot be reconciled with facts established for determining occupational cancer incidence. Whenever the intensity and duration of exposure to an occupational carcinogen increases, there rises the cancer incidence rate among the exposed population group. There is no plausible reason to assume that the inhalation of allegedly carcinogenic tobacco smoke would be exempted from this rule. The complete lack of even minor increase of laryngeal cancer during the past five decades, although the larynx forms a part of the smoke tract, also militates against the tobacco smoking theory of lung cancer.

It may be concluded that the existing evidence neither proves nor strongly indicates that tobacco smoking and especially cigarette smoking, represent a major or even predominating causal factor in the production of cancers of the respiratory tract and are the main reason for the phenomenal increase of pulmonary tumors during recent decades. If excessive smoking actually plays a role in the production of lung cancer, it seems to be a minor one if judged from the evidence on hand. However, it may be well to remember in this connection, the concluding statement of Doll and Kermaway; that "the study of the relation between the national consumption of tobacco and the national incidence of cancer of the lung has scarcely begun."

5. Cancer of the Lung and Its Relation to Specific Agents.

It should be apparent from the preceding discussion that evidence related to large population groups with exposures to diverse environmental agents with mainly unknown carcinogenic properties gives at best information of only circumstantial value, which may serve as a lead for more specific and detailed subsequent survey studies. The establishment of firm causal connections between environmental influences and the development of cancers of the respiratory system therefore, depends upon the demonstration of definite and significant statistical and causal relations between exposures to specific environmental or occupational agents and/or working conditions and the subsequent appearance of cancers of the respiratory system. It is for this reason that at present the only conclusive evidence on the causal relationship between exogenous factors and cancer of the lung is of occupational origin. In fact, the great majority of the occupational cancers discovered during the present century affect the lung and/or other parts of the respiratory tract (nasal cavity, paranasal sinuses, larynx). While exposure to the so far known respiratory carcinogens occurred in all instances by inhalation, the successful production of lung tumors in experimental animals receiving various carcinogenic chemicals (urethane derivatives, acetylaminofluorene, etc.) through other routes suggests that respiratory carcinogenesis may not be limited also in man to contact with environmental carcinogens by inhalation.

The respiratory carcinogens so far recognized differ greatly in their chemical and physical properties as well as in the physicochemical status in which they come in contact with the respiratory tissue. Some are chemical agents such as various metals or metal compounds (arsenic, chromium, nickel) or minerals (asbestos) or aliphatic organic (isopropyl oil) or aromatic organic compounds (chemical carcinogens in coal tar, pitch, soot, petroleum derivatives). Others exert a carcinogenic effect through physical forces (ionizing radiation),

such as the various radioactive chemicals. They enter the respiratory tract in the form of dust, fumes, mists, vapors and gases. Depending upon the particle size of the dispersed carcinogenic material they may penetrate into different parts of the respiratory tract. If the particles are rather large, they produce mainly cancers in the upper parts (nasal cavity, larynx). They may enter, on the other hand, the deeper portions, such as the bronchi and lung or paranasal sinuses, if they are of very small particle size or take the form of vapors or gases.

The depth of penetration of atmospheric contaminants into the respiratory tract depends moreover on the intensity of exposure and on a possible destructive action of the ciliated epithelial lining and its mucus producing glands of the upper respiratory tract (nose and trachea). Whenever under the influence of an overwhelming or excessively prolonged exposure to pollutants of the air the limit of the protective saturation of the nasal and tracheal mucosa is reached, dispersed particles will penetrate thereafter in increased amounts into the deeper portions of the respiratory conduits.

The relative degree of carcinogenic property of the various respiratory carcinogens depend not only upon their intrinsic carcinogenic potency, but also on the degree of dispersion, their solubility in water and fats, and their direct cytotoxic qualities, all of which influence the action of a particular carcinogen by controlling its penetration into cellular elements and its time of retention and possible permanent deposition in the lung tissues. Since under occupational conditions, exposure to respiratory carcinogens is not infrequently complicated by contact with various noncarcinogenic types of dust, solvent vapors, acid fumes and alkali and oily mists, the primary and direct action of the carcinogens may be modified by an interaction with these concomitant agents or with the anatomic reactions set up by these agents in the lung.

Recent studies of Falk and Steiner and of von Haam, Titus, Shinowara and Caplan, have shown that carcinogenic hydrocarbons (3,4-benzpyrene, pyrene, 20-methylcholanthrene), when adsorbed to the surface of carbon particles, such as those forming the bulk of various carbon blacks (commercial soots), are released only with difficulty by elution with solvents and show within a given time limit a lowered carcinogenic potency when tested in their adsorbed form on mice. It may be assumed that similar conditions prevail when carcinogenic hydrocarbons are adsorbed to the carbon particles of ordinary soot or to particles of silica present in road dust or released as waste catalyst from catalytic cracking towers of oil refineries. Counterbalancing this slowed release, however, is the longer retention of the carcinogenic hydrocarbons when introduced into the lung tissues on the surfaces of chemically rather inert, noncarcinogenic particles.

Similar modified conditions of exposure are apt to prevail if radioactive gases adsorbed to the surfaces of noncarcinogenic mine dust enter the lung, and are bound to stay in this adsorbed form over a longer period in the lung and, thereby, exert a more pronounced carcinogenic effect upon the lung tissues than when entering these organs as free radioactive gases (radon, thoron). While a few of the respiratory carcinogens cause the development of pulmonary fibrosis and pneumoconiosis (radioactive dust and gases, asbestos, chromium compounds, beryllium, soot and tar fumes), such effects do not accompany the carcinogenic action of others (nickel, arsenic, isopropyl oil, petroleum products).

Consideration also must be given to the possibility that the simultaneous action of pulmonary fibrosis producing dusts or chemical vapors, gases and fumes associated with, preceding or following the action of specific respiratory carcinogens might interfere with the removal from the lungs of inhaled carcinogenic material due to the obliterations of lymphatics and blood vessels

and the formation of fibrous barriers. Such anatomical complications present in the lung again would most likely tend to prolong the action of inhaled carcinogenic agents, and thereby intensify their specific effect on the lung tissues. On the other hand, there exists adequate evidence for the view that established pulmonary fibrosis would hinder to some extent for similar reasons the spread of a cancerous growth within the lung as well as to other tissues.

Fibrogenic pneumoconiosis thus may exert a definite effect on the productive and developmental phases of pulmonary cancer, as well as on its subsequent proliferative phase.

Consideration must further be given to the concept that carcinogenesis by specific agents may be accelerated by the simultaneous action of nonspecific chronic inflammatory processes. Some investigators have proposed that bronchial cancers originate in scars or mucosal pigment perforations produced by the deposition of inhaled dust (Schmorl):

Finally, pneumoconiotic and fibrotic lesions are clinically important from a differential-diagnostic viewpoint, since some of these changes may resemble neoplastic lesions in their symptomatology or they may obscure the presence of such conditions. From the general evidence available on lung cancer it appears, however, that the great majority of these neoplasms are not associated with pneumoconiotic conditions.

Probst pointed out that pneumoconiosis usually is absent in lung cancer. Bauer stated that the inhalation of stone dust as such certainly does not elicit the development of a pulmonary cancer since there exist many industrial operations with definite pneumoconiotic hazards without an increased liability to lung cancer. In the report of the British Empire Cancer Campaign of 1952 the statement appears that pneumoconiosis was present in only one (0.1%) of 325 cases of pulmonary cancer. Olson reported that in a series of 69 cases seen in Boston, pneumoconiosis was present in only 2.9 percent. However, such

observations obviously disregard the rather frequent occurrence of anthracotic and bituminotic pneumoconiotic deposits in the lungs of persons living in industrialized regions. There exists, moreover, the possibility that benign pneumoconiotic conditions elicited by dust of black metallic compounds such as iron oxide or chromite may be mistaken at autopsy for anthracosis. The differentiation between carbon pigments and those of metallic nature requires the use of spodograms or ashed sections. This procedure, however, is not routinely applied.

A. Relation of Inhalation of Metal Dusts and Fumes and of Metal
Pneumoconiosis to Respiratory Cancer.

The chronic dust disease of the lung (pneumoconiosis) caused by the occupational inhalation of dusts and fumes of metal, metal compounds and metal alloys only exceptionally assumes a progressive, fibrogenic character (beryllium). As a rule, metal pneumoconioses are of the so-called benign type; i.e., they cause non-disabling reactions sometimes associated with minor fibrosis around dust particles (subpleural, peribronchial and perivascular tissues). Such "benign" lesions when severe may ultimately lead to a narrowing of the lumens of bronchi and blood vessels and, thereby, in the long run interfere with the proper blood circulation in the lung causing an embarrassment of the right cardiac function. Massive accumulations of so-called inert dusts, moreover, may cause tissue necrosis and the formation of cavities. The pulmonary reactions occurring after inhalation of dusts, fumes or vapors of some metals are mainly of inflammatory and possibly allergic nature (metal fume fever) (manganese, cadmium, zinc, selenium, copper, brass). Both acute inflammatory and chronic fibrosing reactions may be observed with others (beryllium). The metals and metal compounds which deserve consideration in this respect are aluminum, magnesium, beryllium, cadmium, calcium, copper and copper alloys, vanadium, chromium, manganese, nickel, zinc, tin, antimony, mercury, molybdenum,

lead, iron, tellurium, tungsten, titanium, barium, selenium, thallium, arsenic and silver (Greenburg; Hamlin; Silson; Elkins).

Inhalation exposure to these metals may be related to various occupational activities (mining, smelting, casting, polishing, grinding, drilling, powdering, bagging, alloy making and processing, metalizing, electroplating, soldering, welding, metal catalyst production and use, pigment production and consumption (paints, inks, glazes, glass coloring, textile, paper, rubber, plastic and linoleum dyes), spraying, chemical and metallurgical processing, etc.).

The number of workers who are exposed to the various metallic dusts and fumes in American industries is considerable. Bloomfield, Trasko, Sayers, Page and Peyton produced the following estimates (1940) based on the survey of all industries of 10 states and a total of 1,503,204 workers exposed: Lead: 76,743 workers; antimony: 9,735 workers; chromium: 7,976; arsenic: 3,356; mercury: 3,220; cadmium: 3,031; manganese: 2,287; other metals: 220,929; total: 327,277 (22% of worker population surveyed). From additional estimates made by these investigators it can be assumed that approximately the same percentage of industrially employed workers in the rest of the United States has some form of occupational metal dust exposure.

The epidemiologic information on hand indicates that respiratory cancer hazards definitely exist for only a few of the metal dust and fume exposures, that they are potentially present or controversial for others and that for most of them they do not exist. For many, adequate epidemiologic information is not available. Some metals, however, have produced cancers in organs other than the respiratory ones (liver, bone) of experimental animals. Very defective is the information as to the existence or nonexistence of occupational lung cancer hazards for the various metals, metal compounds, types of exposures and industrial operations in which metals with established respiratory cancer hazards are used and produced.

a. Aluminum.

With enormous growth of the aluminum industry, exposure to aluminum dust has become rather widespread during recent decades for various types of industrial workers. During the past decades, it has been proposed and used on a limited basis as an alleged preventive and therapeutic of silicosis (Brown and van Winkle; Berry; Jephcott and Johnston).

While American investigators are, in general, rather skeptical as to the actual existence of an aluminosis (Crombie, Elaisdell and McPherson; Hunter, Millon, Perry and Thompson; Folicard; Ehrismann; Editorial, Lancet), European investigators have described roentgenologic and pathologic changes of diffuse pulmonary fibrosis occurring in workers occupationally exposed to the inhalation of aluminum dust (propeller grinders, reduction furnace workers, aluminum powder workers, aluminum alloy manufacturers (Goralewski; Goralewski and Jaeger; Koelsch; Jaeger and Jaeger; Modder and Schmitt; Schwallnus and Kleinsorg; Feil; Perry; Filipo; Gerstel; Doese; Kahlau). The recent observations of Wyatt and Riddell on the diffuse pulmonary fibrosis occurring in bauxite workers (Shaver and Riddell; Shaver), tentatively places the blame on the inhalation of amorphous alumina dust or fumes generated during the production of alumina abrasives. It thus may be concluded that a massive and prolonged inhalation of bauxite and possibly also of aluminum oxide dust or fumes may produce fibrotic pulmonary changes (aluminosis).

As to an alleged carcinogenic action of ingested aluminum, first proposed by Odier, it can be stated that such claims have not been confirmed by subsequent investigators (Doese; Bordas; Marie-Amero; Ichok; Bertrand and Serbescue; Elumenthal; Editorial, J.A.M.A.). This negative evidence does not favor the concept of a carcinogenic action of inhaled aluminum dust on the lung tissue, although a final decision of this question must await the result of comprehensive and competent epidemiologic surveys of exposed worker groups for the incidence of respiratory cancers.

b. Magnesium.

The situation as to the inhalation of magnesium dust and its relation to pulmonary neoplasia is similar to that recorded for aluminum. The inhalation of magnesium dust and magnesium oxide fumes in foundries, airplane manufacturing plants, etc. is considered as not particularly harmful although the occurrence of disturbances has been reported (Teleky; Gardner and Delahant; Ford and Stern; Elkins). The absence of reliable and competent surveys of respiratory cancer incidence among magnesium workers does not permit to render at this time any definite opinion as to the existence of a lung cancer hazard. There is, however, no evidence available for suspecting the occurrence of such a complication.

c. Barium.

The development of pneumoconiosis (barytosis) following the inhalation of barium sulfate among workers of barite mills has been reported (Spedini and Valdini; Teleky; Arrigoni). Nothing is known as to the existence of an excessive liability to lung cancer hazards among the exposed workers.

d. Cadmium.

Contact of the respiratory organs with cadmium vapors, fumes or dust occurs during the smelting of cadmium ores, working up of residues, spraying of cadmium containing paints and pigments, welding of alloys, and other industrial operations (Division of Industrial Hygiene, NIH and may give rise to acute pulmonary reactions (edema and hemorrhages; pneumonitis) (Fairhall). The published records do not contain any reference to pulmonary neoplasia as a late effect of prolonged occupational exposure to cadmium fumes, dust and vapors (Greenburg).

e. Calcium.

The deposition of lime dusts in the lungs (calcicosis) of lime stone miners, lime burners, marble polishers, gypsum manufacturers, etc. considered as harmless,

gives rise to disseminated foci of peribronchitis and interstitial pneumonia (Teleky). Causal relations to pulmonary cancer from occupational exposures to lime dust are unknown.

f. Cobalt.

Exposure to cobalt dust especially by cobalt ore miners has figured for many years as an alleged cause of cancer of the lung among the Schneeberg miners (Carozzi; Schinz). The increasing use of cobalt in alloys of steel, as a catalyst in the Fischer-Tropsch process of oil production from coal and in the manufacture of iron free magnets and carbide tools and dies has brought a considerable number of workers in contact with cobalt dust (Fairhall, Keenan and Brinton). Granular or conglomerate markings were found in the lungs of such workers upon radiologic examination (Fairhall, Castberg, Carozzi and Brinton). Although Schinz reported the development of a spindle cell sarcoma of the femur in a rabbit which received an intraosseous implantation of powdered cobalt more than three (3) years previously, the survey conducted in 1925 by a special committee of the Labour Office of the League of Nations among workers employed in cobalt mines in other parts of the world (Canada: Cobalt City; Congo: Katanga; Norway: Skuterud; France: Allemont; Czechoslovakia: Dobschina) failed to show any excessive liability of cobalt miners to lung cancer. German investigators, moreover, noted that the high lung cancer frequency in Schneeberg was limited to miners and was absent among the workers employed in the cobalt pigment factory. It may be mentioned, moreover, that the chemical analysis of the lung of a Schneeberg miner who died with lung cancer did not reveal any cobalt (Beyreuther). The evidence on hand supports the view that a carcinogenic action of cobalt upon the lung tissues has not been established and that claims to that effect are not supported by valid evidence.

g. Titanium.

Since the extensive use of titanium and its compounds in paints, steel alloys, cemented carbides, screening smoke and in protective ointments against flash burns is of rather recent date, relatively little information is available as to the existence and type of pulmonary effects produced by the inhalation of dust of metallic titanium and its compounds, mainly titanium oxide. Carozzi thought titanium oxide to be nontoxic because of its chemical inertia. Elina did not observe any pulmonary reactions among workers exposed to titanium oxide dust. However, guinea pigs exposed to this chemical showed after two (2) months an increase of connective tissue of the lung and abundant exudate in the large and medium sized bronchi. Similar observations were made by Lenz (1936) in guinea pigs exposed to the inhalation of clouds of pure titanium oxide, according to Teleky. It seems that under the circumstances, long range epidemiologic studies are indicated for determining whether or not the inhalation of titanium and its compounds may be associated with any lung cancer hazard.

h. Antimony.

Antimony and its compounds are used in considerable quantities in alloys (lead), type metal, bullet cores, pigments in rubber, enamels, paints and textile dyes, bronzing powders and medicinal agents. An appreciable portion of the workers using antimony and its various compounds are exposed to antimony dust and fumes (type setters, metallurgical workers, rubber workers). Irritative symptoms of the upper respiratory tract including perforation of the nasal septum have been reported to occur among these workers (Feil; Shirley; Schwartz and Tulipan). Fairhall and Hyslop stated that antimony metal dust appears to be much more toxic than the dust of other insoluble antimony compounds of industrial importance. They noted, moreover, in connection with industrial exposure to the various antimony dusts, that finely divided antimony

dust can remain suspended in the air longer than would be anticipated with a heavy metal.

None of the few existing studies of antimony dust hazards in industry apparently paid any attention to the possibility of a lung cancer hazard for workers exposed to these chemicals.

i. Beryllium.

Beryllium is a metal which has found significant industrial use only since about 1920. It was not until about 1940 that beryllium and its compounds were extensively employed for numerous purposes and products (beryllium-copper, beryllium-aluminum and beryllium-nickel alloys, glass, phosphors in fluorescent lamps and neon tubes, atomic energy products, ceramics, refractories, x-ray tube windows; vitreous enamel; radio tubes, textile fibres, gas mantles) (Galman; Gardner; Vorwald; Breslin; Hardy; Hyslop, Palmers, Alford, Monaco and Fairhall). It is evidently for this reason that untoward effects in persons exposed to the inhalation of dusts and fumes of beryllium and its various compounds (beryllium sulphate, beryllium chloride, beryllium oxide, beryllium fluoride, beryllium carbonate, beryllium manganese silicate, beryllium oxy-fluoride, beryllium silicate, beryllium hydroxide, zinc manganese beryllium silicate) have been recognized only during the last decade. These manifestations were of both acute and chronic nature as far as the respiratory organs were concerned (acute beryllium pneumonitis, chronic pneumoconiotic granulomatosis (berylliosis). Some investigators used the term sarcoid in describing the histologically peculiar, pulmonary manifestations (Gardner; Hardy, Vorwald; Eisenbud, Wanta, Dustan, Steadman, Harris and Wolf; Eisenbud, Berghout and Steadman; Corcoran; Sterner and Eisenbud; Machle, Beyer and Tedbrock; Laskin, Turner and Stockinger; Johnstone, Bruce, Lovejoy, Brothers and Velaquez; Higgins; Vigliani; Wilson; Van Ordstrand, Hughes, DeNardi and Carmooy; Aub and Grier; Shilen, Mellor, Koppenhaver, Cleland, Galloway and Lutz; Hasterlik; Jetter; Ginabat; Van Ordstrand, DeNardi and Schneider; Sander; Kline, Inkley and Tritchard; Fenn; Silson, Benjamin and Wilson; Williams;

Martland, Brodtkin and Martland; Wilson; Machle; Dutra; Morgis and Forbes; Reynolds; Hardy, Bartter and Jaffin; Cass; Pomeranz and Brodtkin; Klemperer; Pascucci; Pyre and Oatway).

Hardy noted as a conservative estimate there occurred up to September 1951 between 300 - 400 cases of acute beryllium poisoning and at least 200 cases of the chronic variety in the United States which resulted in the fluorescent-lamp industry in a mortality of about 25 percent among the ill workers. It is remarkable, moreover, that chronic berylliosis has appeared not only among exposed workers, but also among persons living in the neighborhood of fluorescent lamp factories and inhaling their beryllium containing effluents (Eisenbud, Berghout and Steadman; Eisenbud, Wanta, Dustan, Steadman, Harris and Wolf). Similar observations on occupational berylliosis were reported from Germany, Italy, England, Russia and Canada (Hardy; Gerrie, Kennedy and Richardson; Kennedy). Not infrequently similar granulomatous lesions have been observed in other parts of the body after the usually traumatic introduction of beryllium dust, especially of beryllium phosphors from broken fluorescent tubes. The skin of the fingers and hands was the most frequent extrapulmonary location of these reactions (Nash, Grier and Freiman; Helwig; Davis and Grimes; Curtis; Large and Stumpe; Gerrie, Kennedy and Richardson; Dutra; Silverman and Erickson; Stokes, Beerman and Ingraham). Beryllium granulomas have also been found in the nose (Kazanjan and Joseph) and in the anterior ocular structure (Rizzuti),

It is noteworthy that beryllium apparently once inhaled is retained over a long period of time in the human body, since beryllium was detected in the urine up to 10 years after cessation of exposure (Klemperer, Martin and Van Riper) and has been demonstrated in the lungs of rats one year after the inhalation of beryllium oxide (Dutra, Largent, Cholak, Hubbard and Roth) as well as in their bones (Stokinger, Steadman and Root; Barnes) where it may replace calcium. The skeleton retains the bulk of the beryllium in the body (50-80%)

if the inhaled aerosols are soluble compounds, such as beryllium sulfate and beryllium fluoride; the lungs retain the bulk of beryllium if the compounds are insoluble, such as beryllium oxide. Experiments of Aldridge, Barnes and Denz, moreover, have shown that beryllium ions react rapidly with certain tissue proteins and form complexes with plasma proteins when introduced into the blood. These complexes protect the beryllium from being precipitated by phosphate ions.

The metabolic peculiarities of beryllium compounds obtain special importance in view of the fact that Gardner in 1946 reported the production of osteogenic sarcomas in rabbits injected intravenously with insoluble beryllium containing powders (beryllium phosphate, zinc beryllium silicate). Other investigators subsequently confirmed these results with the same and other beryllium compounds (beryllium oxide, beryllium silicate; metallic beryllium) introduced into rabbits by the intravenous or respiratory routes (Sissons; Barnes, Denz and Sissons; Hoagland, Grier and Hood, Nash; Hoagland and Hood; Dutra, Largent and Roth; Barnes). The preparatory period for the sarcomas was from 11 - 24 months. Commenting on the successful production of osteogenic sarcomas in rabbits after inhalation of beryllium oxide, Dutra, Largent and Roth noted the fact also that the bones of persons dying with berylliosis contained not inconsiderable amounts of beryllium. They came to the following conclusions: "During the last 20 years, considerable numbers of persons have been exposed to dusts of poorly soluble compounds of beryllium in various industries throughout the United States. Despite the fact that cases of cancer of this type have not been reported, it is possible that the inhalation of poorly soluble compounds of beryllium may eventuate in osteogenic sarcoma in man, Presumably, the incubation period of such tumors would be considerably longer in man than in rabbits, and observations may be required over a period of years before it will be known whether persons who have been exposed to beryllium

are prone to have such tumors." Barnard also suggested that osteogenic sarcoma from compounds of beryllium "might possibly be another industrial hazard." So far, only rabbits have responded with the development of osteogenic sarcomas following the administration of beryllium compounds. The direct introduction of powdered beryllium metal into the femoral cavity of rats, into their pleural cavity and into the paranasal sinuses failed to elicit a single neoplastic response at the site of injection in any one of the 85 animals used within an observation period of two (2) years (Hueper).

When in 1948 Hueper proposed that the sarcoid pulmonary manifestations of berylliosis might be followed by outright malignant lesions in the lungs, this suggestion was received with a great deal of scepticism. The recently reported successful production of bronchogenic carcinomas in the lungs of rats which inhaled over periods of more than one year, dust of soluble and insoluble beryllium compounds (Vorwald), however, makes the appearance of such delayed malignant sequelae in man a distinct possibility.

In view of the established occupational as well as general environmental occurrence of human berylliosis, it may be pointed out that the discovery and identification of this pneumoconiosis was definitely facilitated by the distinctive and definitive histologic features of the disease. If these manifestations should be followed by the development of cancers of the bones and lungs, the establishment of causal relations between a previous exposure to beryllium and the subsequently appearing cancerous reaction would appear to be a rather easy proposition. The studies on the toxicity and carcinogenicity of beryllium compounds indicate that the toxic and cancerous manifestations are to be considered as responses to the action of beryllium itself and not as the result of the associated anions of its acidic salts. (Stokinger, Sprague, Hall et al.). In considering possible future carcinomatous developments in persons with previous exposure to beryllium, some consideration also may be given to the

toxic effect exerted by beryllium on the liver leading to the development of cirrhosis and to an impairment of the metabolic and detoxicating function of this organ (Aldridge, Barnes and Denz; Hoagland, Grier and Hood).

k. Selenium

Selenium and its compounds have increasingly been employed by industry during the past two (2) to three (3) decades for various purposes (glass decolorizer, production of ruby glass, glazes, paints, pigments, inks, coloring of plastics, steel and copper alloys, rubber accelerators and antioxidants, photoelectric apparatus, fireproofing of electric cables, insecticide chemicals) (Buchan; Manville). Workers engaged in primary and secondary industries handling selenium containing ores (copper, lead, zinc, pyrites, lime and cement) and selenium products may become exposed to the inhalation of dust and fumes containing metallic selenium, selenium oxide and hydrogen selenide (Smith, M.I.; Dudley and Miller; Dudley; Buchan; Moxon and Rhian).

There may occur an environmental exposure to selenium containing dust in those parts of the United States in which selenium fumes are released from metal ores smelters (Montana, California, Tennessee) (Byers) or where a seleniferous soil exists (Wyoming; South Dakota) (Smith, Franks and Westfall). In addition to toxic manifestations in other organs, especially the liver (Lillie and Smith; Cameron; Smith, Stohlman and Lillie; Fitzhugh, Nelson and Eliss) and nervous system, the inhalation of selenium causes respiratory irritation, with cough, sore throat, and pulmonary edema (Greenburg; Motley, Ellis and Ellis).

There is no recorded evidence indicating that industrial and environmental chronic selenosis produces an increased liability to lung cancer in man and animals, although a prolonged oral administration of selenium to rats resulted in the development of liver adenomas and carcinomas (Nelson, Fitzhugh and Calvery) as well as thyroid adenomas (Seifter, Ehrlich, Hudyma and Mueller).

1. Manganese

Manganese is used chiefly in the steel industry, but also in the chemical, ceramic, glass, dye and varnish industries and in electrotechnics. Cases of poisoning may occur whenever manganese is handled and particularly when manganese dust is produced. Manganese enters the organism mainly through the respiratory passages. While the symptoms of chronic manganism are caused by changes in the central nervous system, pneumonia is a rather frequent reaction among manganese workers and has a high mortality rate (Gärtner; Baader; Voss; Joetten and Reploh; Kahlstorf; Gundel and Heine; Joetten, Reploh and Hegemann; Zanetti; Blüttner; Elstad; Blüttner and Lenz; Davies; Ehrismann; Vigliani; Fairhall and Neal). Large and numerous deposits of manganese ore dust were found in the lungs (Schopper; Bauer; Boemke) of manganese workers.

There do not seem to exist any data on the lung cancer incidence among manganese workers.

m. Copper

The industrial use of copper, copper compounds and copper alloys (brass, bronze, etc.) has been very extensive for many years and a great number of workers, therefore, are exposed to the inhalation of copper dust and fumes (mines, smelters, foundries, bronze powder operations, parasite repellent applications on hulls of ships, fungicide and insecticides production and use, plating operations, paint production and use, ink manufacture, etc.).

While inhaled copper dust and fumes may elicit irritative symptoms of the respiratory tract, cuprosis is infrequently mentioned as a cause of benign pneumoconiosis (Query; Schlotz). Ulcerations of nasal membranes and perforation of the nasal septum have exceptionally been observed among workers of copper plating operations (Barsky). It is noteworthy, however, that copper and brass workers have an highly excessive mortality from pulmonary "tuberculosis" (Hayhurst). Since occupational cancers of the lung as well as cryptogenetic

cancers of this organ not infrequently have masqueraded in the past as tuberculosis of the lung, a thorough and competent clinical and epidemiologic investigation of copper, brass and bronze workers of all types is urgently needed for determining lung cancer morbidity and mortality among members of this occupational group. The recent study of Snegireff and Lombard on the cancer incidence among copper smelter workers is too inadequate and methodologically defective to be of any real value.

n. Tin

Tin is extensively used in alloys, bronze, brass, foil, plate and solder.

While tin ores are not mined in the United States, there exist one (1) tin smelter (Texas) and several tin oxide recovery plants.

The inhalation of dust and fumes of tin and tin oxide, especially by furnace tenders, may give rise to the development of a pneumoconiosis (stannosis) which roentgenologically resembles in appearance silicosis and baritosis (Pendergrass and Pryde; Cutter, Faller, Stocklen and Wilson), although anatomically there is an absence of fibrosis (Bartak and Tomecka; Dundon and Hughes). The lungs show a fine network of black lines, which on histologic examination is represented by particulate dark brown and black pigment located in interalveolar septa and peribronchical and perivascular tissue.

There is no information as to the incidence of lung cancer among tin workers. None of the so far reported cases of stannosis had such a neoplasia, although one died from cancer of the prostate.

o. Lead

Lead is used as a metal as well as in the form of various compounds for various industrial purposes (pigment in paints, inks, glazes, and enamels, in the manufacture of storage batteries, antiknock agents (tetraethyl lead), insecticides, glass, cable coverings, ammunition, sheet lead, pipes, etc.). During the mining and smelting of lead ores and during the manufacture, processing and

use of the various products workers become exposed to the inhalation of lead dust, fumes and vapors. Chronic lead poisoning is, in fact, one of the most common occupational diseases.

While chronic plumbism as a rule is not characterized by the deposition of lead in the lung nor by the development of fibrosing pulmonary reactions, Black commented on the fact that cancer of the lung had been observed in workers exposed to the inhalation of lead or lead compounds. In support of a possible causal relation between exposure to lead dust and fumes and cancer of the lung, he cited the occurrence of pulmonary cancer in glass workers (Boyd; Gutzeit; Klotz) and type setters and printers (Seyfarth). Rosedale and McKay noted that nine (9) out of 43 persons with lung cancer had been working more or less with lead in one form or another. Black added two (2) cases of lung cancer (linotype worker, metal polisher) to the previously published number of 15 cases of pulmonary cancer in lead workers. He found a chronic fibrous pneumonia in both of his cases.

Because of the close chemical relation between lead and radium and in view of the occurrence of radioactivity in some lots of commercial lead (due to the presence of radium D) he believes that the pulmonary reactions reported represent results of the inhalation of radioactive lead. These allegations have so far remained unconfirmed. Although it may be advisable to ascertain through epidemiologic surveys of various types of lead workers, whether or not there exists an excessive liability to pulmonary neoplasia, it is not likely that such complications of chronic plumbism exist because of the thorough and prolonged study which chronic lead poisoning has been subjected to by investigators of many countries. The reported excessive frequency of lung cancer among painters who are known to be affected not infrequently with plumbism probably has no causal relation to lead exposure because painters, especially spray painters, inhale also other paint constituents, some of which have known carcinogenic properties (zinc chromate, carbon black, etc.).

p. Zinc

Zinc metal and zinc compounds (zinc chloride, sulfide, oxide) are used industrially on a large scale for galvanizing, brass production, alloys, paint pigments, rot-proofing compounds and sheeting.

Inhalation of zinc dust and fumes occurs during smelting, galvanizing, spraying, brass founding, brazing and welding of galvanized iron operations. Such exposures give rise to attacks of metal fume fever. Lehmann stated that volatilized zinc upon cooling forms zinc oxide which in turn is transformed into zinc carbonyl. This is hygroscopic and when inhaled spreads over the surfaces of the respiratory structures, producing a necrotizing effect on the exposed cells. Exposure to fumes of zinc chloride, which is a known corrosive and occasionally still used in the chemosurgical treatment of skin cancers, has resulted in injury to the mucosa of the nasopharynx and respiratory tract (Evans, E. H.).

Whether or not there exists a chronic type zinc poisoning (duBray; McCord and Friedlander), however, is still controversial (Drinker and Fairhall; Hegsted, McKibbin and Drinker; Nuck, Remy and Holtzmann; Gocher). Although the experiments of Nuck et al. on dogs exposed to the inhalation of zinc dust showed that the zinc content of the lung, liver and kidney decreased rapidly after cessation of exposure, the conclusion that such a rapid removal of zinc from the organism precluded the development of a chronic type of zinc poisoning is scarcely acceptable, since many examples of chronic poisoning can be cited which demonstrate that a prolonged retention of a toxic agent is not a prerequisite for the causation of chronic poisoning provided there were contacts with the offending agent over long periods of time. At any rate, it is a well-established fact that under such conditions of exposure a rapid removal of the environmental agent from the site of contact would not preclude the subsequent development of a cancer,

While there does not exist any evidence indicating that an occupational exposure to zinc dust and fumes creates an increased liability to cancer of the respiratory organs, Cristol reported that zinc is present in increased quantities in all malignant growths and that, therefore, a decision should be reached whether this metal might be a carcinogenic agent. In fact, there exists a fair number of observations on the successful production of testicular teratomas in fowl following the intratesticular injection of zinc chloride (Falin and Gromzewa; Falin; Anissimova; Bagg; Bertrand and Vladesco; Falin and Anissimova; Falin and Gromtseva; Ljvruga; Michalowsky). In view of these findings it seems to be desirable to study population groups occupationally exposed to zinc dust and fumes in regard to lung cancer frequency.

q. Vanadium

Vanadium is one of the group of metals which have obtained rapidly increasing importance during the past one (1) to two (2) decades in metallurgical operations. It is mainly used as an alloy in steel and as a catalyst in the production of diamond black, sulfuric acid, phthalic anhydride and benzoic acid.

Exposure to vanadium dust exists in the mining and smelting of vanadium ores. The smelting process is accompanied by the production of dust of vanadium pentoxide (Symanski). The inhalation of this dust has caused among exposed workers pulmonary dust reticulation as well as chronic rhinitis, and bronchitis (Wyers; Balestra and Molfino).

Apart from the fact that vanadium dust apparently is retained in the lung, thereby causing a chronic metal effect upon the lung tissues, its possible carcinogenic relation is complicated by the fact that vanadium ores in this country contain uranium and other radioactive substances. It is for this reason that vanadium ore miners and smelter workers are not suitable subjects for a study of possible carcinogenic vanadium hazards connected with the

inhalation of vanadium dust and fumes. Such future investigations should be conducted on worker groups whose members are not in contact with any known and recognized environmental carcinogen. At present nothing is known concerning any carcinogenic properties of vanadium and its compounds.

r. Iron

The extensive production and use of various types of iron and of diverse iron products offers frequent opportunities for the inhalation of dust and fumes of iron and its various alloys and compounds (iron ore miners, arc welders, grinders, polishers, silver finishers, metal workers).

The resulting red or black siderosis caused by pulmonary retention of Fe_2O_3 or of $\text{Fe}_3\text{O}_4 \cdot \text{H}_2\text{O}$, respectively, is considered an inert form of pneumoconiosis which does not cause disability and which at least in part seems to be reversible (Greenburg; Pendergrass and Leopold; Jones and Lockhart; Pendergrass, Lane and Ostrum; Doig and McLaughlin; Bohrod; Barrie and Harding; D'Onofrio and Passeri; Sander; Dreessen, Brinton, Keenan, Thomas and Place; Teleky and Gilbert; Humperdinck; Buckell, Garraud, Jupe, McLaughlin and Perry; Balsac and Feil; Lanza; Stewart and Faulds; Boamke; Koelsch; Hamlin; Silson; Teleky). The deposition of iron oxide particles does not elicit in the lungs a progressive and marked fibrosing reaction (Harding, Grout and Davies), unless the inhaled dust contains also silica, producing then a siderosilicosis (Teleky).

The coexistence of siderosis and cancer of the lung has occasionally been observed (Stewart and Faulds, one (1) case; Dreyfus, three (3) cases in watch makers; Vorwald and Karr, three (3) cases in hematite miners; Simons, one (1) case in a blaster of iron casts). It may be mentioned, moreover, that Kennaway and Kennaway reported a 2.25 fold incidence of pulmonary cancer among metal grinders and that Turner and Grace as well as Campbell noted an excessive frequency of lung cancer among metal workers.

Experimental studies on animals exposed to iron oxide and hematite, respectively, gave contradictory results as to the production of lung tumors. While Vorwald and Karr, using guinea pigs and rats failed to obtain lung cancers with hematite dust, Campbell reported an increase in the number of lung tumors in mice exposed to iron oxide over that of the control series,

When the available evidence is viewed critically, it is not likely that exposure to iron dust conveys an abnormal liability to the development of lung cancer. The excessive frequency of this malignant neoplasm among metal workers in general may well be due to exposure to dusts, fumes and vapors of specific carcinogenic metals, such as chromium, nickel, arsenic, and perhaps beryllium, or to mists of lubricating and cutting oils. It may be noted, however, that Warren and Drake when discussing the causation of primary hepatic carcinoma in livers with hemochromatosis stated "Together, primary progressive parenchymal damage to the liver and subsequent deposition of iron in hepatic cells seem to provide a setting for carcinogenesis more effective than simple portal cirrhosis." Whether similar considerations might be applicable to siderosis and cancer of the lung seems to be doubtful in view of the few cases reported so far, in which the two conditions co-existed. However, thorough and comprehensive epidemiologic data on the incidence of lung cancer in workers exposed to iron dust are not available and a definite conclusion on this problem, therefore, must be withheld. The availability of conclusive information on this point appears to be urgent, since damages have been allowed in the past by court action in at least one case of cancer of the lung in the production or aggravation of which the inhalation of steel dust was alleged to have played a significant role (Medicolegal Abstracts).

s. Nickel

Nickel, one of the most industrially important metals, and principally mined in the Sudbury district of Ontario, Canada, finds many uses (alloys

(iron, copper, chromium, aluminum, cobalt), molybdenum) employed in the manufacture of stainless steel, heat resisting steels, forgings, casts, wires, sheets, structural shapes, tubing, rods, bars, strips, etc.), electroplating, catalysts, ceramic enamels and colors, pigments in paints and inks, storage batteries, etc. (International Nickel Co.; Friend; Davis).

Exposure to nickel fumes and nickel dust of metallic nickel and its compounds or to nickel carbonyl vapors is, therefore, frequent for industrial workers of many types and in many operations (Sappington; Fischer). While skin contact to nickel and nickel salts not infrequently results in the development of an apparently allergic type of dermatitis (Burckhardt; Wedroff; Weber; Schittenhelm and Stockinger; Müllschitzky; Kolzoff; Cormia and Stewart; etc.), inhalation of the volatile nickel carbonyl has been responsible for an appreciable number of acute and often fatal poisonings (Armit; Amor; Mott; Brezina; Kötzing; Brandeis; Bayer; Krafft; Goldblatt and Wagstaff; Drinker, Fairhall, Ray and Drinker; Hamilton; Royer). The pulmonary manifestations (congestion, desquamation of alveolar epithelium, fibrinous acellular exudation into alveolar spaces, bronchial mucosal hemorrhages) are apparently attributable to the toxic action of finely dispersed nickel formed from the disintegration of nickel carbonyl upon the pulmonary structures (Henderson and Haggard). Krafft suggested that these reactions are the result of a nickel allergy having the lung as its shock organ.

The first report concerning the occurrence of an excessive number of cancers of the nasal passages (nasal cavity and paranasal sinuses) and of the lungs among workers on the Clydach plant of the International Nickel Company located at South Wales, England, was made by Grenfell in 1932, although the first appearance of these neoplasms among the nickel refinery workers was noticed in 1924 (Baader). Subsequent reports dealing with these cancers were made by Stephens; Amor; Cooper; Carozzi; Bridge; and Merewether. From 1923 to 1948 inclusive, there were notified with the Chief Inspector of Factories

a total of 47 cases of cancer of the nose and 82 cases of cancer of the lung, from the nickel works. By the end of 1948, 46 of the nasal cancer and 72 of the lung cases had died. None of the nasal cases and only two (2) of the lung cases had commenced work in the nickel refinery after 1924 when a reconstruction of the plant had been carried out. The average exposure period for the nasal cancers was 23 years (range, 3-26 years), and for the lung cancers 25 years (range, 1-33 years). No cases of cancer of the larynx have occurred, and only one (1) cancer of the nasopharynx was observed at Clydach.

The nasal cancers involved the turbinates, nasal septum and paranasal sinuses (ethmoids). Of these the majority were of the undifferentiated cell type (6), some showed a squamous cell character (3), while columnar cell carcinomas were uncommon (1). Of the lung cancers, of which histologic studies were available in only four (4) cases, three (3) were of the small cell, pleomorphic type, while one (1) was a squamous cell carcinoma.

Similar observations were recently recorded from a Norwegian nickel refinery, where three (3) cases of lung cancer were seen (Løken). In one of these cases a squamous cell carcinoma was associated with sarcoid lesions,

Goldblatt and Wagstaff mentioned that so far cancers of the respiratory tract have not been noted among the workers employed at the German nickel refinery at Ludwigshafen, nor has there been observed an unusual frequency of respiratory cancers among the workers of the Sudbury nickel ore mines and smelters in Canada.

Amor pointed out that the majority of individuals employed at Clydach who developed respiratory cancers were not exposed to the inhalation of nickel carbonyl but to that of nickel matte dust or dust from the nickel matte roaster (Løken). More recent data communicated by Morgan confirmed this observation, although exposure to nickel carbonyl vapors had occurred more frequently among the affected workers than apparent from the data previously

given by Amor. The relatively high incidence of cancer of the nasal cavity indeed suggests that a rather coarse particulate dust readily arrested at the region of the turbinates may have been active in the production of cancers at this particular site, while nickel containing vapors or a very small particulate dust most likely account for the cancers of the lung and nasal sinuses.

As to the causative agent, various theories have been advanced. Amor favored the concept that the inhalation of mist from arsenic containing sulfuric acid used in the refining process was the active carcinogenic agent. It is most unlikely that this is correct because the nickel refinery workers do not suffer from perforated nasal septa and display no evidence of chronic arsenicism such as dermatosis and cutaneous cancers, which almost always have accompanied the occurrence of lung cancer among workers exposed to arsenical dusts or fumes (Hueper).

Amor stated that the refined nickel-copper ores are free from radioactive matter. The respiratory cancers observed among nickel refinery workers thus are not identical in etiology with those seen in miners employed in the radioactive mines of Schneeberg and Joachimsthal.

Workers employed at the roasters, in the nickel carbonyl operation and at other parts of the plant, on the other hand, become exposed to the inhalation of dust, fumes, or vapors containing nickel. Nickel is the common denominator for all of them. It thus is most probable that the respiratory carcinomas observed among nickel refinery workers are reaction products to more or less finely dispersed nickel particles or vapors. There is no evidence available, however, which indicates that the inhalation of nickel in particulate or vaporized form is accompanied by pulmonary changes of a pneumoconiotic nature.

The concept of a nickel etiology of respiratory cancers was tested in animals by Campbell, who exposed mice to the inhalation of powdered nickel

matte and observed that these animals had a lung tumor incidence significantly higher than that of the unexposed control mice. The recent experiments of Hueper seem to demonstrate more conclusively the carcinogenic properties of metallic nickel. When pure metallic nickel powder was implanted into the femoral and pleural cavities of rats, cancers developed at the site of injection within six (6) to 24 months in about 40 percent of the surviving rats. Among 14 tumors thus produced, 13 were sarcomas and one (1) was a squamous cell carcinoma.

Whether or not nickel assumes a carcinogenic role for cancers of other organs and following exposures by other routes is unknown. It may be mentioned, however, that Araki and Mure demonstrated by spectrographic methods, nickel in human and animal cancers of various types and sites. The nickel content ranged from 6.273 mg. per kg. of fresh tumor tissue to 0.2 mg./kg.

No assessment of the degree of occupational nickel cancer hazard can be made from the data available, since the number of workers at risk is unknown. Likewise, no definite opinion can be expressed as to the possible existence and extent of respiratory cancer hazards for persons having for other reasons contact with dust, fumes and vapors containing nickel or its compounds.

t. Chromium

Chromium as a metal, alloy or compound is used for many purposes in industry. It is for this reason that a large number and variety of workers have contact with chromium and chromium compounds and that even restricted groups of the general population may possibly become exposed to these agents in the form of dust, vapor, fumes, mist, liquid and solid (Bourne and Ruskin). Workers most likely to be exposed to chromium and its compounds are acetylene workers, aniline workers, bleachers, blue printers, chrome workers, chromium platers, chromate manufacturers, chromite miners, crayon makers, dye workers, electroplaters, enamel workers, glass and pottery frosters, glass colorers,

pottery glazers, artificial flower makers, battery makers, linoleum workers, paint makers, ink makers, painters, photographic workers, photoengravers, polishers, printers, rubber workers, steel workers, tannery workers, vulcanizers, waterproofers of textiles and paper, welders, users of chromate anti-rust agents (locomotive attendants and engineers), bitumen and oil refinery workers (U. S. Department of Labor; Sappington; Abraham).

An environmental atmospheric contamination with chromium compounds may result from the release of chromium containing industrial wastes of chromate plants and of oil refineries using a chromium containing silica catalyst for the catalytic cracking of oils. An environmental spread of chromates may also follow the use of such compounds as anti-rusting agents in automobiles. Since many of the industrially used chromium compounds exert a corrosive action on tissues, skin contact and/or inhalation of such agents results in the development of chrome ulcers of the skin and nasal septum, which in turn provide definite proof of an existing health hazard. Commenting on the appearance of such manifestations among workers of new industries using chromium compounds, the Chief Inspector of Factories of England and Wales remarked in his report of 1944 that "the control of old hazards in new industries is of interest to others as well as to the student of industrial health, for it would seem that in many cases the hazard is not recognized until damage to tissue has been done, when old principles have to be relearned and adapted to new uses."

This reflective observation seems to be quite appropriate when contemplating the possible existence of respiratory cancer hazards for individuals employed in the numerous industrial operations in which chromium and its compounds are handled and for which no pertinent published data of any kind exist at the present time (Chief Inspector of Factories, 1947, 1948; Regional and Field Letter OFS; Engelhardt and Mayer; Mayers; Schwartz and Dunn;

Leroux-Robert; Bloomfield and Blum; Foil; Galloro; Akatsuka and Fairhall; Schrapf; O'Donovan; Deribere; Vaccero; Goldman and Karotkin; Mancuso; Manciola; Buess; Winston and Walsh; Harrold, Meek, Collins and Markell; Edmundson; Lieberman; Dixon; Carter; Peroni; Galloro).

The observation of apparently occupation-connected cancers of the respiratory organs, especially the lung, has been limited so far to two types of operations, the production of chromates from chromite ore and the manufacture of certain chromium pigments (zinc chromate, barium chromate, lead chromate). In these operations both water soluble and insoluble chromium compounds are inhaled by the exposed workers. The chemical nature of the actual carcinogenic agent which is responsible for the excessive liability of chromate and chromium color workers to cancer of the lung is still controversial. Although all investigators believe that some chromium compound or compounds are causally involved, it has remained uncertain whether the compounds suspected are hexavalent or trivalent, water soluble or insoluble, monochromates or dichromates. Water soluble chromium compounds (monochromates, dichromates, and zinc chromate) are most often incriminated. Mancuso and Hueper recently pointed out that it may be more likely that carcinogenic effects are elicited by chromium compounds which are either not soluble in water or only slightly so, because such chemicals when inhaled as dust would be retained and deposited in the lung and thus exert a prolonged effect upon the pulmonary tissues. Such chromium compounds present in a chromate plant would be represented by chromite ore and its early conversion products preceding the formation of monochromates. These little water soluble trivalent chromium compounds occur in the material present in mixers and roasters and are contained in the slag which usually is stored for future use in the yard area of the plants.

Supporting this concept as to the chemical nature of the carcinogenic chromium compounds is the fact that workers as well as animals exposed to the

inhalation of chromite ore dust have not only a high chromium content of the lungs but also an excessive blood chromium level (Mancuso and Urone). Recent experiments on rats which inhaled finely powdered chromite ore dust showed that after 18 months a chromium level of 13.0 and 17.0 gamma, respectively, in 100 cc of blood was found in two (2) rats studied. This finding, moreover, definitely establishes the fact that a fraction of the chromium contained in chromite ore is solubilized in the pulmonary tissues and discharged into the blood.

Additional support of a causal role of trivalent compounds may be derived from the observation that 10 workers of the 20 chromate workers with lung cancer reported on by Alwens and Jonas in 1938, were not employed within the manufacturing buildings or were repair men or maintenance workers (blacksmith, glazier, drivers, welder, or manufacturers of sulfuric and hydrochloric acid, produced in a nearby building). While all of them probably had some exposure to chromates, it is likely that their contact with chromite ore dust or with dust from the slag heaps containing more or less "insoluble" chromium compounds was much more pronounced (Mancuso; Urone, Druschel and Anders; Bourne and Yee; Buchell and Harvey).

As the result of the retention of "insoluble" chromium compounds in the lung tissues, there develops a blackish spotty pigmentation and a spotty fibrous thickening of the peribronchial and interstitial tissue where the chromium dust particles are deposited. This pneumoconiotic condition called chromitosis was described by Andrievskaya and Mislavskaya in chromite ore miners and by Lukanin; Letterer, Neidhardt and Klett; and by Mancuso and Hueper in chromate manufacturers. It was produced experimentally in rabbits by Lukanin. Letterer reported a chrome-silicosis in a polisher of an iron foundry who inhaled silica and chromium oxide dust.

While the attempts of Gross and Koelsch; and Campbell to produce lung cancer in mice by their exposure to chromate dust were unsuccessful, Schinz and Vollmann, who implanted powdered chromium metal into the femoral cavity of rabbits observed after more than three (3) years, one (1) animal with cancer of the lung and one with cancer of the femur. In a series of pilot experiments recently conducted, guinea pigs, rats and mice were exposed to the inhalation of chromite ore dust at a concentration of 155 gamma per liter of air for five (5) to six (6) hours per day, four (4) days per week, for a total of 18 months, three (3) of the six (6) Wistar rats showed tumors involving respiratory organs. One (1) rat had a squamous cell carcinoma originating in the paranasal sinuses and invading and dislocating the eyeball, two (2) additional rats showed at autopsy, sarcomatous growths involving the mediastinal lymph-nodes and lungs. There was much black pigment within the thickened interstitial tissue. The lungs of three (3) guinea pigs revealed on histologic examination a marked proliferation of the bronchiolar epithelium which often occluded the bronchiolar lumina by the formation of cellular plugs consisting of oval and spindle shaped, hyperchromatic epithelial cells. These cellular masses extended in places into the the adjacent alveolar lumens forming polyps, coatings or solid casts. These changes were widely distributed in the lungs which showed, moreover, a scattering of dark brown to black amorphous pigment located especially in the subpleural zone and within a fibrous and thickened interstitial tissue.

These multicentric proliferative lesions of the bronchiolar epithelium resemble in many respects those recently described by Spain and Parsonnet in a woman and termed by them "minute bronchiolargenic carcinomas." Although the carcinomatous as well as sarcomatous reactions observed in rats and the hyperplastic bronchiolar lesions found in guinea pigs provide suggestive evidence that a prolonged inhalation and pulmonary retention of chromite

dust may have a cancerigenic effect upon pulmonary tissues, this evidence is at present far from being conclusive.

It is definitely surprising that an excessive liability to lung cancer has been established so far only for chromate workers in Germany (Pfeil; Alwens and Jonas; Teleky; Carozzi; Gross and Koelsch; Alwens, Bauke and Jonas; Lehmann; Martineck; Gross; Koelsch; Alwens, Bauke and Alwens; Goldblatt and Wagstaff) and United States (Machle and Gregorius; Gregorius; Baetjer; Hueper, Mancuso and Hueper; Imprescia) and in chrome pigment workers in Germany (Baader; Gross and Koelsch; Letterer, Neidhardt and Klett). Bidstrup found a single case of lung cancer upon x-ray examination of the chest of 321 chromate manufacturers employed for more than 10 years in English plants, while no data exist on this point in regard to chromate producing or consuming plants in other countries, such as Switzerland, Italy and France (Wackman; Saita; Lecoœur).

Apart from the excessive frequency of lung cancers among chromate workers which according to American observations range from 13 to 31 times the normal, an occupational origin of these cancers is strongly suggested by the shift of the age distribution toward younger age groups. This is particularly striking for the lung cancers present among German chrome pigment workers, since 50 percent of the cancers affected individuals before the age of 40 years when lung cancers of unknown etiology are relatively infrequent. (Table 10).

The quantitative data on the chromium content of various organs and blood of persons with chromium lung cancer have been reported by several investigators (Alwens and Jonas; Jonas; Letterer, Neidhardt and Klett; Mancuso and Hueper), Spannagel recently noted a peculiar behaviour of the chromium content of the blood and urine in chromate workers before and after the development of lung cancer. It was found that the normal urinary excretion of chromium ceases in workers with the development of lung cancer

- 61a -

Table 10.

Age Distribution of Chromium Cancers of the Lung

| Years | 21-30 | 31-40 | 41-50 | 51-60 | 61-70 | 71-80 | Total |
|--------------------------|-------|-------|-------|-------|-------|-------|-------|
| Cases: American Chromate | 0 | 8 | 16 | 19 | 10 | 1 | 54 |
| German Chromate | 1 | 3 | 7 | 14 | 12 | 1 | 38 |
| German Chrome Pigment | | 5 | 3 | 1 | 1 | | |

while simultaneously the blood chromium level becomes elevated. If confirmed, this observation may have distinct importance in causal, metabolic and diagnostic respects.

With the exception of two (2) cases (one (1) cancer of the nares (Newman); one (1) cancer of the maxillary sinus (Goldblatt and Wagstaff), the lung was the exclusive site of respiratory cancers observed among chromate workers. The total number of these cancers is at present around 125 cases from all sources. None has been reported as originating from nasal septum ulcers.

Chromium pneumoconiosis thus seems to accompany the development of cancer of the lung in chromate and chrome pigment manufacturers. It is uncertain, however, whether the pneumoconiotic process plays an essential or modifying role in the specific cancerization process or whether it is merely a phenomenon of coincidental coexistence.

u. Arsenic

Arsenicals represent a by-product or waste product of the smelting of many ores (copper, zinc, silver, cobalt, antimony, iron, bismuth, nickel, tin and lead). Arsenicals are present in the smelter fumes and slag heaps. They are extensively produced and used, especially during past decades, as insecticides, fungicides and vermicides (sheep and cattle dip, grasshopper bait, rat poison) as well as a herbicide, especially for clearing railroad rights-of-way. Arsenicals are applied as sprays to orchards and vineyards and are dusted from airplanes upon cotton, corn, soybean and potato fields. They are employed as wood preservatives, in the manufacture of glass, lead-base alloys, dyestuffs, bronzing and paint pigments, and medicinal and cosmetic preparations (Mote). Arsenic and its compounds constitute, according to "Environment and Health", a health hazard for 35,251 workers employed in American industries. This definitely is a very conservative estimate of the

number of exposed workers considering the long although incomplete, list of different occupations entailing contact with arsenicals given by Chamberlain. The estimate, moreover, does not include the rather considerable number of persons who are exposed to arsenicals for purely environmental reasons by either ingesting arsenicals with foodstuffs contaminated with arsenical insecticide residues or consuming drinking water polluted with arsenicals leached into drinking water supplies from mine and smelter dumps or inhaling arsenicals released into the air from industrial establishments or by small or large scale dusting operations of arsenical pesticides.

From the published evidence on hand, it appears that environmental and non-occupational contacts with arsenicals have been responsible in recent decades for the majority of cases of chronic arsenicism and cutaneous arsenical cancers (Neubauer; Hueper; Arguello, Tello, Macola and Manzano; Butzengeiger; Beader; Nieberle; Hofmann; Prell; Holmquist; Montgomery and Waisman; Cannon; Arhelger and Kremen; Straube; Bohnenkamp; Hanser and Simon; Gonnet; and many others).

While the causal role which arsenic plays in the production of cancers of the skin on the basis of chronic arsenicism of occupational, medicinal or environmental origin has long since been firmly established, it is of rather recent date that exposure to arsenicals has seriously been considered as a principal causal agent of cancers of the mucous membranes, such as those of the bronchi, stomach and bladder. Indeed, today there exists as yet only highly suggestive but not conclusive evidence linking cancer of the lung with an occupational exposure to arsenical dust. However, in almost all cases of lung cancer for which such claims were made, there existed stigmata of chronic arsenicism in the form of arsenic dermatosis with or without skin cancers. Since the inhalation of arsenical dust and fumes produces rather frequently the development of perforated nasal septa as well as chronic irritative

conditions of the bronchi, thereby creating a symptomatic cancerigenic pattern similar to that seen in chromate workers, the existence of a causal relationship between cancer of the lung and chronic arsenicism appears under such circumstances to be a reasonable conclusion. Chest and x-ray examinations of forty (40) workers employed in an arsenic smelter revealed a mild degree of pneumoconiosis (Saupe).

Although Saupe himself did not discover any evidence of lung cancer among the workers studied - even though they often were afflicted by hyperkeratoses of the skin and perforated nasal septa - he cited the autopsy observations previously made by Schmorl on two (2) arsenic smelter workers who died with cancer of the lung (Teleky). Frommel briefly mentioned the occurrence of a cancer of the lung in a taxidermist who used an arsenical powder for dusting the pelts of animals. Four (4) additional cases of lung cancer in sheep dip workers with arsenic dermatosis noted in one (1) of these were reported by Merewether, while Hopkins and Van Studdiford observed in a farmer living near a cotton field sprayed with insecticides, arsenical dermatosis, epitheliomas and cancer of the lung. The occurrence of five (5) cases of lung cancer (Merewether; Hopkins and Van Studdiford) among only 24 individuals suffering from occupational arsenical dermatosis and epitheliomas caused Neubauer to wonder whether this is mere coincidence, because only two (2) cases of lung cancer were observed among 143 cases of medicinal arsenic cancers of the skin (Russell and Klaber), or under occupational conditions whether the irritation of the respiratory tract by arsenical dust was responsible for the phenomenon.

Henry commenting on the occurrence of skin cancers among sheep dip workers (1910 - 1923) recorded the occurrence of two (2) additional cases of lung cancers among ten (10) workers of this group having cutaneous cancers. He mentioned, moreover, the presence of cancers of the left foot, abdominal

wall and lung in a furnaceman of a sodium arsenite factory. Analyzing the mortality experience of a sheep dip factory, Hill and Fanning found that seven (7) or 31.8 percent of the 22 cancers causing death among members of this group were located in the respiratory organs, while three (3) or 13.6 percent were situated in the skin. There were during the period 1910 - 1949 a total of 75 deaths from all causes. The proportional excess of cancer deaths was mainly attributable to an excessive frequency of cancers of the lung and skin which were confined to workers in the chemical processes and were absent among members of the general group who would be unlikely to be exposed to any specific hazard. Perry, Bowler, Buskell, Druett and Schilling concluded from the clinical evidence obtained that after many years of exposure to arsenicals these sheep dip workers may develop a squamous cell carcinoma in the bronchus.

The most recent addition to epidemiologic investigations on arsenic cancer was made by Snegireff and Lombard studying cancer deaths among employees of several metallurgical plants of unidentified type. Of the total of 109 deaths from all causes recorded during the last 25 years, twelve (12) were due to cancer of all sites and of these, six (6) were located in the lungs. The investigators concluded from this evidence that "there are indications that biologically the human race made the adjustment to arsenic in the environment and that only rarely, when associated with other contributing endogenous factors such as systemic disease, or possibly factors such as radiation, it may be capable of upsetting the biological equilibrium"; and further "that the handling of arsenic trioxide in the industry studies does not produce a significant change in the cancer mortality of the plant employees; hence other factors in addition to arsenic must be considered significant in the causal relationship to cancer". In view of the fact that 50 percent of all cancer deaths among employees of one (1) plant surveyed were

caused by cancer of the lung, the observations made in fact confirm a carcinogenic action of inhaled arsenic trioxide upon the tissues of the lung of the exposed workers. The conclusions of the authors seem to be based on wishful thinking and not on a rational evaluation of facts. This interpretation of the data of Snegireff and Lombard is supported by the high incidence of lung cancer among the population of several counties in Montana where copper smelters and mines were operated for many years creating an occupational and environmental pollution of the atmosphere and soil with arsenicals. Prolonged inhalation of arsenical dust and fumes appears to produce an increased liability to cancer of the lung.

However, the existence of such connections should be acknowledged only when there existed at some time, clinical and, if possible, histo-and biochemical evidence of chronic arsenicism. In view of the absence of any such evidence associated with chronic arsenicism among the nickel refinery workers affected by cancers of the nasal cavity, paranasal sinuses and lung, among excessive tobacco smokers with cancer of the larynx and lung, it is most unlikely that exposure to arsenic dust, fumes and vapors plays any role in the production of respiratory cancers in members of these population groups.

Comments on Respiratory Metal Cancers

The evidence presented demonstrates that some metals and metal compounds when inhaled as dusts, fumes, mists or vapors are capable of producing cancers in various parts of the respiratory tract. Pneumoconiotic changes precede and accompany such developments when chromium compounds are inhaled, while such lesions have not been reported following the inhalation of nickel compounds and are apparently of minor nature after the inhalation of arsenicals. Although the respiratory introduction of beryllium compounds leads in animals to the development of progressive granulomatous pneumoconiotic alterations which may become complicated in rats by bronchiogenic carcinomatous manifestations, no such malignant sequelae has been reported as yet in human berylliosis.

Whether or not respiratory metal cancers may develop after the inhalation of other metals represents a problem deserving serious consideration especially where such metals are widely used or have attained major industrial importance only in recent years.

B. Relation of Inhalation of Silicon and Carbon Polymers to Respiratory Cancer

The second group of respiratory carcinogens is composed of chemical agents of rather diverse composition but sharing the characteristic that they may form or occur as linear polymers. The principal evidence suggesting the existence of "polymer cancers" has come from experimental observations. The "so far" known or suspected carcinogenic polymers are either polymerized carbon compounds or silicon compounds (Table 11).

Cancers of the respiratory tract in man seem to be causally related to both varieties of polymer carcinogens.

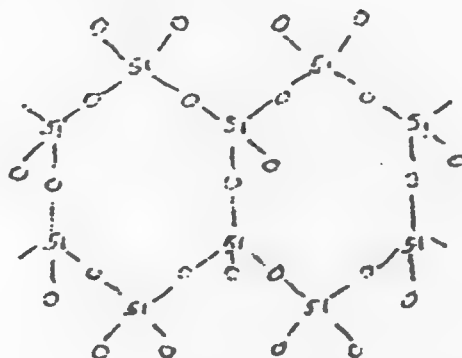
a. Carbon Polymers.

The first experimental evidence apparently representing a carcinogenic effect of a carbon polymer was provided by Turner who obtained in four (4) of nine (9) rats sarcomas at the site of subcutaneously implanted bakelite discs. Bakelite is a phenolformaldehyde polymer. More recently Oppenheimer, Oppenheimer and Stout succeeded in obtaining sarcomas in rats and mice after inbedding subcutaneously various plastic films, namely, cellophane, alcohol-extracted cellophane, a commercial polyethylene, a very pure polyethylene and vinyl chloride film. Sarcomatous reactions were absent following the subcutaneous implantation of linters, surgical cotton, and glass cover slips. Druckrey confirmed these findings on rats which received subcutaneous and intraperitoneal implants of cellophane and of cellophane extracted with boiling benzene. Sarcomas were also obtained by Druckrey in rats which were intraperitoneally injected with a polyamid polymer, namely, ϵ -caprolactam.

CHAIN AND BAND STRUCTURES OF CANCERIGENIC POLYMERIZED CHEMICALS

I. ASBESTOS; FIBROUS POLYMERIZED SILICATE

CHAIN
STRUCTURE -

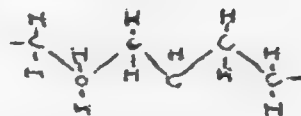


BAND
STRUCTURE -

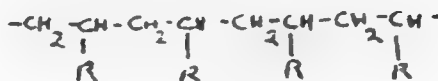


II. CARBON POLYMERS

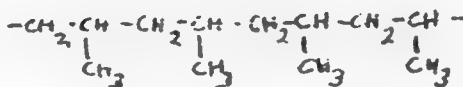
A. POLYETHYLENE - POLYTHENE (POLYETHYLENE FILM)



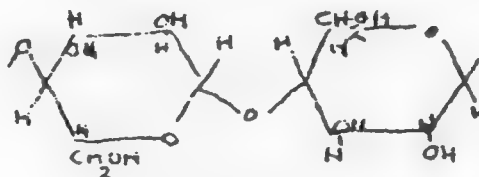
B. POLYVINYL (POLYVINYL FILM)



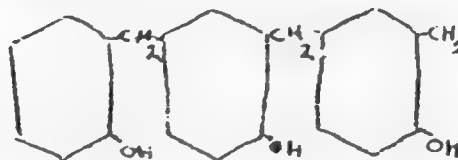
C. POLYPROPYLENE (POLYPROPYLE OIL?)



D. CELLULOSE (CELLULOSE)



E. PHENOL-FORMAL- DEHYDE RESIN (Bakelite)



Negative results, on the other hand, were noted with Orlen, Nylon and Perlon as well as glass wool.

Additional experimental evidence supporting a cancerigenic role of linear polymers can be deduced from the observation that certain methylolamines, ethyleisimines and epoxides, which readily form linear polymers, possess cytotoxic (canceroclastic) and cancerigenic properties (Elmore, Gulland, Jordan and Taylor; Goldacre, Loveless and Ross; Philips; Hendry, Rose and Walpole; Hendry, Homer, Rose and Walpole).

Several other industrially important polymers, such as synthetic rubbers, polystyrenes, polymethacrylates, urea-formaldehyde and melamine-formaldehyde resins have as yet not been tested for potential cancerigenic properties.

Through the recent discovery of cancers of the paranasal sinuses, larynx and lung among isopropanol manufacturers, the occurrence of carbon-polymer cancers has probably been extended to man. Isopropyl oil, i.e., the crude liquid from which isopropyl alcohol is distilled and which is a slightly turbid, viscous liquid, turning, upon standing, slowly into a brownish to blackish tarry material, contains polypropylene compounds as well as propylene ether which may be oxidized into propylene peroxide and propylene epoxide having a tendency to polymerize. Polypropylene, merchandised as Opponol K, is used commercially as an oil for cable filling (Schildknecht).

Workers employed in isopropanol manufacture have been exposed to the inhalation of vapors, mist and dust of isopropyl oil, escaping from leaky pipe connections, defective pumps and gaskets or spilled on the floor at the occurrence of breaks in pipelines and during repairs on pipes, pumps and stills. Weil, Smyth and Nale reported that between 1928 and 1950, a total of seven (7) neoplasms affecting various parts of the respiratory tract (nasal sinuses (4), larynx (2), and lung (1) came to observation among 71 employees or in 8.4 percent who worked more than five (5) years in the isopropanol plant. Inhalation

experiments conducted on mice with the various chemicals to which the workers become exposed provided suggestive evidence incriminating isopropyl oil. Since the occurrence of an excessive number of respiratory cancers among isopropanol manufacturers has not been limited to one particular plant, it can be concluded that isopropyl oil, and thus probably polypropylene compounds, represent a human carcinogen.

In an attempt to explain the carcinogenic action of these carbon polymers, Druckrey suggested that they may become bound to cellular proteins by covalence bindings or that low grade polymers may even be incorporated into protein molecules and, thereby, give rise to abnormal proteins, which in turn would be equivalents to the carcinogen-protein complexes established for carcinogenic aromatic amino- and azo-compounds as well as for 3,4-benzpyrene and certain metals (arsenic, chromium, nickel) possessing pronounced allergenic qualities. Perhaps a part of this general reaction mechanism may be represented by the recently reported occurrence of a so-called polymer-fume fever, similar in symptomatology to metal fume fever which in turn resembles anaphylactic shock (Koelsch; Harris).

b. Silicon Polymers

Among the various crystalline, amorphous or colloidal silicates (quartz, asbestos, talc, soapstone, tripoli, diatomaceous earth, silicon carbides, etc.), both asbestos and crystalline silica when inhaled produce specific types of pneumoconioses which in turn allegedly convey an excessive liability to cancer of the lung. Certain silicon polymers thus may display carcinogenic properties similar to those displayed by aliphatic and aromatic hydrocarbon polymers.

I. Silicosis

Since silicotic pneumoconiosis fulfills all the requirements which may be asked from a nonspecific carcinogenic agent acting on the basis of the chronic irritation theory of carcinogenesis, uncomplicated silicotic conditions

anthracosis

classified as

(anthracosilicosis, siderosilicosis, etc.) have repeatedly been claimed as favoring the development of lung cancer (Fine and Jaso; Dible; Charr; Klotz and Simpson; Klotz; Schmorl; Andersen and Dible; Rüttner). Isolated cases or small series of cases showing the coexistence of silicosis with cancer of the lung were reported by Allen; Cramer; Dible; Fine and Jaso; Frommel; Harris; Klotz and Simpson; Maxwell; Middleton; Olson; Sladden; Saupe; Sweany, Porsche and Douglass; Charr; Gsell; Pancoast and Pendergrass; Vorwald and Karr; Homburger). The total number of such combinations reported from this country and abroad is around 75 cases.

Merewether recently added data of a large series to this evidence. Among 6,884 cases of silicosis which came to autopsy, there were 91 cases associated with cancer of the lung (1.32%), which is practically identical with the incidence rate of lung cancer in the general population. Gloyne, however, obtained on several special groups of workers distinctly higher figures. There were four (4) lung cancers among 78 iron and steel workers (5.1%), nineteen (19) lung cancers among 340 pottery workers (5.6%), nineteen (19) lung cancers among 293 coal miners (6.5%), and eight (8) lung cancers among 90 stone masons (8.9%), who showed pneumoconiosis at necropsy. Strieck, on the other hand, did not see any lung cancers among 186 cases of chalcosis, and Böhme recorded the absence of an increased lung cancer rate among silicotics.

Other investigators, also, have pointed out that cancer of the lung is not more frequent or less frequent among workers exposed to the inhalation of silica dust (anthracite miners, pottery workers, sandstone workers, etc.) than among the general population (Vorwald and Karr; Schulte; Schulz; Saupe; Pancoast and Pendergrass; Olson; Fail; Berblinger). Schulte remarked that the pneumoconiosis present in the coal miners of the Ruhr district seems to provide a poor soil for the development of lung cancer. In view of the fibroblastic proliferation stimulating action of silica, Gardner suggested that if anything, there should be an excess of pulmonary sarcomas, but these have not been reported. Since

in this country alone, over one (1) million workers are exposed to silica dust (Environment and Health) and in view of the fact that silicosis among anthracite and metal ore miners (copper, zinc, silver, gold, etc.) still occurs rather frequently, the available evidence supporting a causal relation between silicosis and cancer of the lung is unimpressive.

A lung cancer once established in a pneumoconiotic lung, according to Allen, on the other hand, is not impeded in its pulmonary as well as metastatic spread by the existing fibrotic blockage of the lymph and blood vessels. It seems that this medicolegally important statement needs further confirmation by other investigators, because it is in disagreement with the observations made and concepts held concerning the retarding influence of organ and stroma fibrosis on the proliferative and metastasizing activities of malignant growths in general.

It may be mentioned in this connection that the inhalation of silica dust and the development of lung cancer by the cobalt ore miners in Schneeberg, Saxony, represents only an apparent exception (Schulz) to the negative conclusions reached. It is not the silicosis of the miners' lungs which is of significance but the possible adsorption of radioactive solids or gases to the silica particles which may be of importance in the production of the lung cancers, because such combinations are likely to prolong the carcinogenic action of the radioactive material upon the lung tissues.

Attempts to produce lung tumors in animals experimentally by the inhalation of silica dust (Vorwald and Karr (guinea pigs, rabbits, rats, chickens, mice, cats); Campbell (mice)) gave essentially negative results. Willis and Brutsaert observed in guinea pigs exposed to silicon carbide after 18 to 31 months multifocal proliferations of the bronchiolar epithelium which seem to invade the adjacent alveoli as small dark cell groups. Since adenomatoid structures have been observed in the lungs of apparently "normal" guinea pigs (Sternberg; Leitch), the significance of Willis and Brutsaert's findings is

remains in doubt.

II. Asbestosis

Asbestos differs from the ordinary giant molecular crystalline silicates not only in its chemical and physical properties, but also in the anatomical aspects of the pneumoconiosis produced by it. In contrast to the tridimensional polymerized silica crystals in which no oxygen atoms are left carrying charges to attract positive ions, asbestos consists of giant fibrous molecules composed of polymerized silico-oxygen tetrahedra which are arranged in chains or bands (Parkes). Depending on the origin of asbestos the fibrils may be short or long. Italian, South African and Australian asbestos (amphibols) consists of fibrillar or radiating crystals of calcium-magnesium silicate or sodium iron silicate (40 percent iron oxide). Canadian, Russian, German and French asbestos is hydrated magnesium silicate, which contains small amounts of iron oxide (5.75 percent) Beintker; Wyers; Kruger, Rostoski and Saupe; Vorwald, Durkan and Platt). Canada furnished about 75 percent of the world production of asbestos. Canadian asbestos, because of its long fibers, is especially suitable for textiles.

Depending on its physical characteristics asbestos finds numerous uses (textiles, filter material, building material, gaskets, insulating material, adsorbants, etc.). Some 35,000 workers are exposed in the United States to asbestos dust (Environment and Health).

It is asserted that inhaled asbestos dust produces asbestosis only if the inhaled fibers are sufficiently long. In the absence of a fibrous structure the dust is said to be inert (Wyers; Vorwald, Durkan and Pratt). Since the larger fibrils are arrested in the bronchioles (Gardner), the granulomatous reactions form peribronchiolar fibrous cuffs with giant cells and asbestos bodies. These have a fibrillar core and an iron staining proteninic or colloidal silicic acid sheath. Whether the iron in the sheaths originates

from the asbestos fibers (Timmermans) or is derived from blood or tissue elements (Vorwald, Durkan and Pratt) is still controversial. These two observations deserve special mention because of the apparent dependence of cancerous changes in the lungs of asbestos workers upon the presence of asbestosis and in view of the possibility that the proteins of the lung tissue may specifically interreact with free groups of the filamentary asbestos molecules (Druckrey).

The coexistence of asbestos with cancer of the lung was first reported by Lynch and Smith in 1935 (one case), who recorded later three (3) additional cases (Lynch and Smith; Lynch). Similar observations have subsequently been recorded from this country (Stoll, Bass and Angrist (one case); Holleb and Angrist (two cases); Homburger (three cases)); Canada (Desmeules, Rousseau, Gilroux and Sircis (two cases); Cartier (four cases); Rousseau (one case)); England (Gloyne (17 cases); Harrison (three cases); Merewether (eleven cases); Cureton (one case); Owen (one case)); Germany (Nordmann (two cases); Linzbach and Wädler (one case); Horning (one case); Welz (two cases); Bohne (one case); Domenici (two cases); Baader (one case)). There is, thus, at present a total of 60 cases of asbestosis cancer of the lung on record. To this number must perhaps be added the eight cases of cancer of the lung complicated by asbestosis which Kennaway and Kennaway discovered in an analysis of the death certificates of males registered between 1921 and 1938.

Merewether noted that the mean age of males with asbestosis cancer of the lung was 55.2 years (range 22-72) and that their mean exposure time was 20.1 years (range 6-40), while the mean age of female cases was 44.6 years (range 32-71) and their mean exposure time was 7.6 years (range 0.5 to 48). However, in many cases there elapsed a long exposure free interval ranging from several months to 20 years before the lung cancer became manifest (Wedler).

There were 37 males and 15 females among the 52 cases for which information on sex was available. The male:female ratio is thus 2.5:1, which represents a

Table 12.

| Age Distribution of Asbestosis Cancer of the Lung | | | | | | |
|---|-------|-------|-------|-------|-------|-------|
| Years | 25-34 | 35-44 | 45-54 | 55-64 | 65-75 | Total |
| Cases | 2 | 11 | 16 | 17 | 5 | 51 |

Since lung cancer of unknown etiology occurs rather infrequently before the age of 40, while 26 percent of the asbestos cancers appeared before the age of 44, it seems that there exists a moderate shift toward younger age groups for cancers associated with asbestosis of the lung (table 12).

Table 13.

Exposure Time Distribution for Asbestosis Lung Cancers
Excluding Series of Merewether

| Years | 1-3 | 4-10 | 11-20 | 21 and over | total |
|-------|-----|------|-------|-------------|-------|
| Cases | 4 | 6 | 4 | 7 | 21 |

The exposure time of this series covers a wide range (1-23 years), indicating that type and intensity of exposure to asbestos as well as perhaps an individual susceptibility to asbestosis play an important role in determining the development of this pneumoconiosis and thereby the possibility of a secondary carcinomatous sequela in the lung; (table 13).

marked shift toward the female side when compared with the usual sex ratio of 5:1 to 10:1 for lung cancers of unknown etiology. Equalization of carcinogenic exposure as represented by asbestosis, for the two sexes, thus resulted in a trend toward equalization of liability to lung cancer.

It is of importance to note that the mean age of noncomplicated cases of asbestosis (128) was only 44.2 years (Merewether). One may conclude from this observation that some of these individuals apparently died from asbestosis before their lung cancer had a chance to develop (Linzbach and Wedler).

Additional support for a causal relation between asbestosis and cancer of the lung is derived from the fact that Merewether found among 266 cases of asbestosis observed from 1924 - 1946, 31 cases of coexisting cancer of the lung (13.2 percent), while there were 91 cases of lung cancer with an average age of 59.4 years among 6,884 cases of silicosis (1.32 percent), which came to autopsy. Wedler noted that asbestosis cancer of the lung occurred in 14 cases or 16 percent of 92 cases of asbestosis on which necropsies were performed, whereas the normal rate of lung cancer in autopsy material was estimated to be 2 - 6 percent.

While Lanza, Vorwald, Warren and Cartier are quite sceptical as to the actual existence of an excessive liability of individuals with asbestosis to lung cancer and while Cureton and Homburger are undecided on this question, other investigators favor this concept or consider the existence of a causal relation as highly probable or established (Kennaway; Merewether; Teleky; Nordmann; Hueper; Gross; Lecoeur; Smith, S. W.; Saita; Wegelin; Linzbach and Wedler; Stoll, Bass and Angrist; Welz).

The histologic types of lung cancers observed do not essentially deviate in their relative frequency from those seen in cancers of unknown etiology. There were 22 squamous carcinomas, seven (7) oat cell carcinomas, four (4) anaplastic carcinomas and six (6) adenocarcinomas. In view of the fact that

one of the Norwegian cases of nickel cancer of the lung was associated with pulmonary sarcoidosis, it may be mentioned that Skavlem and Ritterhoff reported the combination of an asbestosis with a sarcoidosis of the lung which, however, was not complicated by a carcinoma.

Attempts have been made to refute the claim of a causal relation between asbestosis and lung cancer by determining the frequency of pulmonary cancer among the total worker population of the asbestos industry (Cartier; Vorwald). Such a procedure is bound to give misleading results. It is quite immaterial how many workers employed in the industry develop lung cancer, since an undetermined portion of these workers doubtlessly sustains either no exposure or only a low intensity exposure and thus does not develop asbestosis of the lung which is the prerequisite for the subsequent cancerous development. Asbestosis must be considered as the essential stigma of an effective exposure. It is, moreover, necessary to know the sex and age distribution of the worker population studied and evaluated as well as the duration of employment and exposure. A marked labor turnover in the industry is not conducive for obtaining reliable information on the actual number of lung cancers and asbestosis cases which may result from effective exposures. For these reasons, no definite conclusions can be drawn from the observation of Cartier, noting eight (8) cases of lung cancer among 4,000 workers studied for 10 years, especially as the frequency of asbestosis among effectively exposed workers increases with the duration of exposure (Böhme; Skull). Kennaway and Kennaway reported that eight (8) lung cancers may be found among 4,000 males of the age range 45 to 64 years.

The evidence on hand, at any rate, has convinced the West German government to make asbestosis cancer of the lung a compensable disease (Tabershaw). Asbestotic warts of the skin, on the other hand, are not covered (Oliver, Morand and Brun; Young; Derwitz; Dreessen, et al.).

The experimental approach to the problem has so far given equivocal results. Vorwald and Karr using guinea pigs which were exposed to asbestos dust obtained negative results. Nordmann and Sorge employed mice for this purpose and claimed to have produced bronchiogenic carcinomas with pulmonary fibrosis in two (2) mice. This observation needs to be confirmed before it can be accepted.

Comments on Respiratory Polymer Cancers

While the fundamental concept of "polymer cancers" is a tentative one and needs to be supported by additional evidence, the available data are sufficiently important to require serious attention from both a scientific and practical viewpoint. The rapidly expanding industrial production and industrial and general use of natural and synthetic polymerized substances in plastics, films, rubbers, textiles, etc., brings a considerable part of the working population in direct contact with chemicals of this type. It seems to be advisable, therefore, to study these population groups during the coming decades for the occurrence of cancers, particularly those affecting the respiratory system.

C. Relation of Inhalation of Combustion and Distillation Products of Coal and Petroleum to Respiratory Cancer

The third major group of recognized, suspected, or potential occupational respiratory carcinogens is composed of substances containing specific carcinogenic aromatic hydrocarbons, and being derived from the incomplete combustion, distillation, catalytic cracking and hydrogenation of coal, petroleum, shale, natural gas, and similar carbonaceous matter. The carcinogenic hydrocarbons are especially contained in the higher boiling fractions of coal tar, pitch, creosote oil, anthracene oil, soot, carbon black, fuel oils, lubricating oils, and crude paraffin oils. The inhalation of dusts and mists of these materials results in their partial retention in the lung and in the production of

anthracosis, bituminosis or chronic oil pneumonia.

1. Coal

Severe and prolonged exposures to carbon dust lead to the development of a mildly fibrosing anthracosis even if not complicated by silicosis (Hollman). Some 650,000 workers in this country are exposed to carbon dust.

The inhalation of coal dust, which is mainly carbon without hydrocarbons, is not credited, in general, as a possible cause of cancer of the lung. A low incidence of pulmonary malignancy (38-71) and laryngeal cancer (44-58) (general population 100) was found to be present in coal miners in England and Wales by Kennaway and Kennaway. This observation agrees with similar ones previously mentioned and covering coal miners in Pennsylvania (Allen), in the Ruhr District of Germany (Schulte; Schul), and in Saxony (Gerbe). Gloyne reported, on the other hand, an incidence rate of 6.5 percent of lung cancer with pneumoconiosis (19 cases) among 293 coal miners who came to autopsy. Gough observed a case of lung cancer in one of six (6) coal trimmers dying from anthracosis after an occupational exposure to coal dust for 25 years,

No information is available on the lung cancer incidence among graphite miners and workers inhaling graphite dust and developing a fibrosing, granulomatous type of pneumoconiosis (graphitosis) which shows a tendency to cavitation (Jaffe; Dunner and Bagnall; Dunner; MacMahon; Harding and Oliver; Dassanayake; Ray, King and Harrison). Graphite is one of the allotropic forms of crystalline carbon which is mined in Ceylon and Ontario, and industrially used in lubricants, polishes, electrodes, crucibles, furnaces, lead pencils and electric batteries.

b. Combustion and Distillation Products of Coal

The apparent innocuousness of coal and, possibly graphite dust as respiratory carcinogens, however, is not shared by the incomplete combustion, distillation and hydrogenation products of coal (pitch, tar, soot, creosote oils, anthracene oils, tar oils, and the highly viscous oily and tarry fractions

obtained by the direct hydrogenation of coal employed by the Bergius process). The carcinogenic action of these combustion and distillation products of coal on man and experimental animals has been established beyond any doubt (Henry; Ross; Hieger; Kennaway; Legge; Cook, Hewett and Hieger; Downing; Earle; O'Donovan; Passey; Pott; Woglom; Twort; Teutschlaender; Oliver; Seelig and Cooper; Goulden and Tipler; and many others). Although the bulk of the Casuistic and epidemiologic human evidence of occupational coal tar and pitch cancers has come from England and Germany, it can not justly be assumed that American made coal tars, tar oils, creosote oils and pitches differ fundamentally in their carcinogenic properties from those manufactured abroad. The exposures sustained by the numerous types of American workers in a great variety of occupations and operations do not seem to differ according to our own observations from those found for their European colleagues, nor are the carcinogenic effects on the skin of these workers at variance with European observations.

However, in addition to skin contact with these products of processed coal, there exists for some groups of workers a considerable exposure to these agents in the form of dust or fumes (tar distilleries, tar paint, shingle, roofing paper, paper conduit, and battery case manufacture, gas works, coke oven operations, road construction and repair work, roofing, brick making, foundries, furnace attendance, railway engine driving, round house operations, pickling of lumber, chimney sweeping, cork brick manufacture, etc.).

Since the high boiling fractions of synthetic oils produced by the direct hydrogenation of coal through the Bergius process have been shown to be highly carcinogenic to the skin and/or subcutaneous tissue of mice and rats, respectively, certain types of workers manufacturing and using such products, inhaling fumes or mists of these carcinogenic petroleum and tar oil substitutes, may have a special lung cancer hazard. Manufacturing plants using the

Bergius process have been operative for some 20 years in Germany and have recently been constructed by several industrial concerns in the United States.

Not only the environmental, but to a greater degree, also, the occupational inhalation of dust, soot and fumes produced by the incomplete combustion of coal results in the development of a soot lung, called bituminosis, which is characterized by the deposition of finely dispersed carbon particles contaminated with hydrocarbons normally contained in coal tar in the interstitial lung tissue (Hübner; Gartner and Brauss). Roentgenologic changes may appear in the lungs after many years of exposure to high concentrations of soot in the air inhaled. While the pulmonary deposition of small to moderate amounts of soot in the lungs, such as commonly found in inhabitants of industrialized regions, does not elicit any appreciable fibrous proliferations, massive storage of soot particles in the lung tissues may finally be associated with an increase of the interstitial connective tissue and with pseudoglandular formations of peribronchial alveoli.

The human evidence relating exposure to coal tar dust and fume with an increased liability to cancer of the lung is equivocal. Kennaway and Kennaway stated that "coal tar in the atmosphere, whether derived from roads, domestic chimneys, or any other source, does not cause an exceptionally high incidence of cancer of the lung." A similar statement was made by Hugounenq and by Husted and Billmann in regard to the liability to cancer of the lung for workers employed in the tar industry and in the construction and maintenance of tarred roads. McLaughlin did not find any lung cancer among 3,959 foundry workers subjected to clinical and x-ray examinations, although there were three (3) deaths from lung cancer among 64 deaths of all causes. Menz recently reported that of 93 workers in Swiss gas plants who died during the 1926 to 1946 period, 21 or 22.6 percent died from cancer of all sites, thereby confirming previous English experience that workers of tar and pitch operations have an excessive liability to cancer in general. Isolated observations

of lung cancer in workers exposed to the inhalation of tar fumes were made by Koelsch (blacksmith, tar worker), Rodenacker (briquette factory worker), Mullschitzky (tar worker).

This predominantly negative information contrasts sharply with observations made among Japanese generator gas oven workers employed in steel plants and among gashouse retort workers in Canada (Kawahata; Kuroda and Kawahata; Cruickshank). The Japanese investigators found within a six (6) year period, 21 cases of lung cancer among generator oven workers who were exposed to the inhalation of hot tar fumes when stoking the coal. An excessive lung cancer rate was absent among workers employed in other parts of the steel mills. The general incidence of lung cancer among the generator gas workers was five (5) per 1,000 workers employed. Seven (7) of these 21 lung cancers occurred in workers 40 years or younger (33 percent, against 18 percent, in cryptogenetic lung cancers) (Hueper). The exposure time varied from nine (9) years to 23 years, the average being 16.6 years. Similar observations were recently made among Canadian gashouse workers. Of 14 cases of cancer among retort house workers, six (6) were due to cancer of the lung, one (1) to cancer of the larynx and one (1) to cancer of the ethmoid sinuses (57 percent cancers of the upper and lower respiratory tract).

It is likely that similar lung cancer incidence rates may exist among American tar workers. Following a visit with a tar distillery where, among the 300 workers during an eight (8) year period, some 25 skin cancers and more than 80 pitch warts had been observed, there was found one (1) case of lung cancer. Subsequent inquiries made by company officials brought the number of lung cancers in this and other tar operations to six (6) cases of cancer of the lung.

From the evidence available it appears that the inhalation of tar fumes sustained by workers of certain operations (coke ovens, generator gas plants, gas plants, tar distilleries) seem to have an excessive liability to cancer of the respiratory tract. It is not unlikely that a more thorough and

competent analysis of the death records of other worker groups, which have so far been found to lack such tendencies, might extend the types and number of tar and pitch workers having an abnormally high respiratory cancer rate.

c. Petroleum, Shale Oil and Natural Gas

The carcinogenicity of certain high boiling fractions of petroleum and oil shale as well as of the combustion products of some of these petroleum derivatives, oil shale and natural gas, have definitely been demonstrated not only on experimental animals but also on workers developing cancers of the skin after prolonged contact with these agents (Leitch; Brockbank and Stopford; Scott; Southam; Twort and Fulton; Twort and Twort; Volkmann; Smith, Sunderland and Sugiyura; Woodhouse and Irwin; Cruikshank and Squire; Auld; Henry; Schamberg; Hayhurst; Heller; Haagensen; and others). Known carcinogenic chemicals, moreover, have been isolated from these petroleum derivatives as well as their combustion products (Berenblum and Schoental; Fischer, Priestley, Eby, Wanless and Rehner; Falk, Steiner, Goldfein, Breslow and Hykes; Waller; Rehner).

In addition to skin contact with carcinogenic petroleum derivatives many workers are also exposed for occupational reasons to an inhalation of oil mist or fumes (workers in paraffin pressing operations, certain groups of oil refinery workers, spinners, metal lathe workers, foundry workers, metallurgical workers, printers, etc.). In spite of this established occupational respiratory exposure to petroleum and shale oils, there is not a single case of oil pneumonia among such workers on record, although such conditions have rather frequently been observed after repeated medicinal instillations of mineral oil containing nasal drops (oil aspiration pneumonitis or paraffinoma of the lung) (Schneider; Rossier and Buhlmann; Kaplan; Ikeda; Graef; Gaertner; Cannon and Wash; Cannon; Bodmer and Kallos; Young, Applebaum and

Wasserman; Berg and Burford; and others). In fact, two (2) cases of cancer of the lung apparently developing on the basis of a medicinal mineral oil pneumonia have been described (Wood; Sante).

The occupational evidence available or published on this aspect is rather scanty and in part controversial. Kennaway and Kennaway found a relatively high ratio of laryngeal, but not of pulmonary cancer in mulespinners, who inhale a mist of the carcinogenic shale oil lubricating the spindles. Southam noted that mulespinners occasionally develop multiple primary cancers involving the stomach or the lung in addition to cancers of the skin. Scott, on the other hand, stated that he had not observed a single case of lung cancer among shale oil workers.

Huguenin, Fauvet and Bourdin, who analyzed a series of 112 lung cancers for possible etiologic factors, found that 18 or 16 percent were metallurgical workers exposed to the inhalation of nebulized lubricating and cutting oils, eight (8) were chauffeurs, five (5) mechanics and one (1) was an engineer. They concluded that their observations indicated an excessively high frequency of lung cancer among workers exposed to vaporized or nebulized lubricating oil. While the study of Gafafer and Sitgreaves on cancer morbidity and mortality among the male employees of an oil refining company did not reveal any abnormal liability of the members of the occupational group to cancer of the lung, this judgment may have to be revised at least for certain types of refinery workers according to more recent and scrutinizing observations. Roesch observed three (3) primary cancers (skin, stomach and lung) in a paraffin worker. Touraine and Bour also attributed the development of pulmonary cancer among certain worker groups to lubricating oil mists. Such exposure conditions may account also for the excessive lung cancer mortality among male metal grinders observed by Turner and Grace.

Since soot as a waste or commercial product has been found to be carcinogenic and to contain known carcinogenic hydrocarbons, a thorough and competent survey

of occupational groups particularly exposed to the inhalation of soot (operating railroad personnel, stokers, carbon black manufacturers, rubber, paint and ink makers, painters, soot burners, printers, Diesel engine drivers, electrode manufacturers, smudge pot operators, phonographic record makers) (Gallie) is an urgent necessity. The negative conclusions reached by Ingalls as the result of a survey of the carbon black industry are based on evidence of dubious merits, because only 79 of the 677 evaluated workers have been employed for 10 years or more in the industry. Since the majority of known occupational lung cancers have an average latent period of over 10 years, his conclusions are actually based on 79 living and active workers. It stands to reason that an analysis of the death records of former carbon black workers may have told a different story, especially if the diagnoses were based on autopsy findings. Such investigations would also add to our knowledge as to the existence, extent and type of bituminosis which might be expected to exist in workers inhaling finely dispersed soot particles. (.

Comments on Respiratory Aromatic Hydrocarbons

At the present time, exposure to tar, pitch, asphalt, heavy fuel oils, lubricating and cutting oils, soot from domestic furnaces, incinerators, industrial power plants, oil refineries, steel plants, metal smelters, carbon black factories, oil dumps, and smudge pots, as well as to the effluents of Diesel and gasoline engines represents the most widespread occupational and environmental contact with carcinogenic material. The specific carcinogenic agents contained in these carbonaceous matters are certain specific aromatic hydrocarbons, which not infrequently are attached to carbon particles giving rise when inhaled to bituminosis or anthracosis or they are constituents of oily matter which when inhaled and retained in the lungs cause oil pneumonia or paraffinoma of the lung.

Since pure anthracosis is not causally related to cancer of the lung, the pneumoconioses accompanying respiratory carcinogenesis by aromatic

hydrocarbons do not play a primary and essential role in this process, although the pneumoconioses may lower the intensity and prolong the duration of the effect of the specific carcinogenic chemicals on the lung tissues.

Since our civilization and economical life has been built around the production and use of the basic carbonaceous substances and their derivatives, it does not seem feasible to attain complete protection against exposure to these carcinogenic chemicals with the preventive and prophylactic engineering and sanitary measures practical and economical at the present time. There is, however, no doubt that a great deal remains to be done in this respect and that we are still rather far-removed from having the maximal amount of possible reduction in exposure to the respiratory cancer producing hydrocarbons contained in the various carbonaceous substances mentioned.

D. Relation of Inhalation of Radioactive Agents to Respiratory Cancer

Up to some ten years ago, occupational exposure to radioactive agents was limited to relatively small groups of industrial and professional workers (miners and refiners of radioactive ores, industrial and medical consumers of radioactive substances (gas mantle manufacturers, luminous dial painters, radio tube makers, physicists and their assistants, radiologists and their assistants). Since the advent of successful atomic fission and the ready production of synthetic radioactive substances the number and variety of individuals who have occupational contact with radioactive matter have rapidly and greatly increased (uranium and thorium ore miners, smelters and refinery workers, atomic energy plant employees, military personnel, agricultural, biologic, medical, chemical, metallurgic, oil, pharmaceutical and other industrial research workers employing radioactive isotopes as well as operators handling directly or indirectly such materials or technical devices giving off ionizing radiation (radioactive static eliminators (Silson; Berman and Ernest; Bryan and Silverman), sewage disposal workers, paper and textile manufacturers, etc.).

It is an established fact that cancers of the skin, connective tissue, bone and blood forming organs have resulted from excessive exposures to radioactive substances affecting the organism or parts of it by various routes. There exists a great deal of highly suggestive, if not conclusive epidemiologic and experimental evidence relating an occupational inhalation of radioactive dust and gasses to the development of pulmonary cancers. Although excessive medicinal and occupational exposure to ionizing radiation (radium, x-radiation) alone may produce in man and experimental animals a fibrosis of the lungs (Kalbfleisch; Doenecke; Belt; Bergmann and Graham; Engelstad; Warren and Gates; Leach, Farrow, Foot and Wawro; McIntosh; Warren and Spencer; Widmann; Bauer; Bauer and Schraer; Tönges and Kalbfleisch; Freid and Goldberg), occupational exposure to radioactive dust and gases has often been complicated by simultaneous inhalation of dust containing various metals (chromium, nickel, iron, arsenic, cobalt) as well as silica. Pulmonary cancers observed among radioactive ore miners, therefore, have been complicated in an appreciable number of cases by silicosis of a minor to moderate degree.

It is for these reasons that the radioactive genesis of the cancers of the lung noted among these miners as well as among uranium and radium refinery workers has been doubted by some investigators, who felt that one of the various nonradioactive metals or the silicosis represented the main causal or an important contributory agent (Schinz; Lorenz; Schmorl; Rostoski and Saupe) or that the available evidence did not provide absolute proof of a radioactive genesis (Lacassagne). Several investigators felt that the lung cancers among the radioactive ore miners in Schneeberg and Joachimsthal were principally attributable to a hereditary predisposition created by inbreeding of the mining population (Macklin and Macklin; Lorenz; Vesin).

The "mala metallorum" causing death at an early age of the miners in the ore mountains of Saxony was first described by Agricola during the early part

of the sixteenth century, and was, subsequently, mentioned by other investigators (Henckel; Scheffler; Thiele). However, it was not until 1879 that its malignant neoplastic character was correctly recognized (Haerting and Hesse). This judgment was, subsequently, confirmed by Cohnhein; Aucke; Arnstein; Uhlig; Risel; Schmorl; Beyreuther; Rostoski, Saupe and Schmorl; Lange; Neitzel; Döhnert; Baader; Teleky; Hueck; Rostoski and Saupe and Schmorl; Thiele; Weber; Koelsch; Lindemann; Doubrow; Brandt; Brezina. Although the miners of the uranium ore mines in Joachimsthal (Czechoslovakia) also were suffering from a fatal lung disease similar to that observed among the cobalt ore miners in Schneeberg, Saxony, it was not until 1926 that the cancerous nature of the pulmonary disease among these miners was recognized (Löwy). Additional confirming evidence was provided later by Beutel; Ziel; Sikl; Saupe; Peller; Pirchan and Sikl; Baader; Behounek and Fort.

Evidence supporting a radioactive origin of the lung cancers among these two groups of miners was provided by the observation of lung cancers among employees of radium refineries and radium laboratories. Löwy reported the occurrence of two (2) such cases among the workers employed in the laboratories of the Joachimsthal mines, where the ores are refined and the purified material is tested. One of the cases had chronic radiodermatitis, leukemia and lung cancer. A similar observation was recorded by Teleky and Neitzel in a German technician of a radium laboratory. The cancerous lung was found to be radioactive. Four (4) cases of lung cancer have recently been observed, according to Baader, among the workers employed in the radium ore processing plant in Belgium, where the occurrence of such complication were previously said to be absent (Maisin, citing Delaet). Perhaps the development of a bilateral alveolar carcinoma of the lung in a woman 16 years after the intravenous injection of 75 cc. of thorotrast may also supply suggestive evidence by the medicinal use of radioactive material arrested in the lung. Mention may also be made in this connection of a report of Martland relating the

occurrence of a cancer of the ethmoid cells in a luminous dial painter, because dial painters not only ingested radioactive material which became deposited in the bones and produced there the development of osteogenic sarcomas, but they also inhaled this matter which, thus, may have produced the carcinoma of the paranasal sinus.

The four (4) cases of cancer of the lung recently reported in an industrial population at an atomic pile site, however, are definitely not causally related to any specific radioactive exposures sustained by the workers concerned because of an insufficiently long exposure and latent period (Love). The argument that these workers were, in part, not directly concerned with radioactive material carries, on the other hand, little weight, as they had, doubtlessly, at times, environmental contact with such material when the meteorologic conditions were unfavorable for the ready dispersal of radioactive wastes at this particular operation.

In favor of an occupational and radioactive origin of the lung cancers among the Schneeberg and Joachimsthal miners is, moreover, the fact that the excessive liability to pulmonary neoplasia is limited to the workers employed underground and is absent among the workers employed above ground, as well as among the population at large of Schneeberg and Joachimsthal, including the employees of the cobalt pigment plant using the Schneeberg ores (Bauer; Schmerl). An excessive lung cancer attack rate, also, has not been found among the miners of the nearby Johann Georgenstadt region where the mines have a low radioactivity. There is, furthermore, no valid evidence on record that miners of arsenic, chromium, nickel, and bismuth containing ores are affected by lung cancers at a rate even remotely approaching that seen among the two (2) radioactive ore miner groups.

The attack rate of lung cancer among the Schneeberg miners has consistently been between 75 and 80 percent since 1879, while that of the Joachimsthal

miners has been stated to range from 10 to 50 percent, although this incidence rate may be too low considering the recent statement of Baader who noted that during the period 1939 to 1943 a total of 180 cases of lung cancer were acknowledged as compensable occupational diseases and that in 1929 there were only 323 miners employed at Joachimsthal. Considering the fact that the exposure and latent period of lung cancer in Joachimsthal miners ranges from 13 to 23 years, it may justly be assumed that these lung cancer cases originated in a miner population of approximately 300 to 400 members working at these mines between 1920 and 1930. The exposure and latent period at Schneeberg is stated to vary from 15 to 18 years for the majority of the cases, but to be occasionally as short as seven (7) years (Baader; Rajewsky, Schraub and Kahlau).

The total number of Schneeberg miners who have died from cancer of the lung between 1879 and 1939, according to available records, stands at approximately 400, while the number of Joachimsthal miners who fell victim to this disease has reached 225 (1926 - 1943). An appreciable number of these miners died at a relatively early age from lung cancer as evident from the data given in table 14, showing the definite shift toward younger age groups.

Measurements of the radioactivity of the Schneeberg and Joachimsthal mines have demonstrated that in both places, mine air and dust have an excessive degree of radioactivity surpassing many times the maximal tolerance dose (Joachimsthal 30 times (Peller)) (Behounek; Behounek and Fort; Tschelnitz; Ludewig and Lorensen; Lange; Rajewsky; Stocklasa). Humphris suggested that the recent introduction of pneumatic drills into these mining operations aggravated the hazard by increasing the production of fine particulate dust containing solid radium.

Repeated attempts have been made to produce cancers of the respiratory tract in experimental animals exposed to the inhalation of radium emanation and/or radioactive mine dust (Schmidtman; Lowy; Campbell; Dohnert; Kahlau;

Rajewsky, Schraub and Kahlau). Schmidtman obtained neither pneumoconiosis nor pulmonary cancer in animals exposed for two (2) years to the inhalation of Schneeberg mine dust collected from drill holes. Campbell, on the other hand, reported that mice inhaling dust of Czechoslovak pitchblende displayed a significantly increased number of pulmonary tumors. In experiments of Dohnert and Hueck, mice were placed in cages within the mines. Some mice developed moderate chalicosis, while pulmonary and mediastinal tumors (adenomas, round cell sarcomas) in addition to an occasional squamous cell metaplasia of the alveolar epithelium were seen in an "abnormally" high percentage of the exposed animals. However, the actual number of affected animals was small, and the interpretation of the results as to their significance, therefore, difficult. Kahlau and Rajewski, Schraub and Kahlau subjected mice to the inhalation of radon. Many of the animals developed bronchial lesions characterized by an atypical epithelial lining as well as pulmonary adenomas (in seven (7) of 12 mice of the test series against one (1) in the control series). While they concluded from this evidence that the radioactive origin of lung cancers in Schneeberg and Joachimsthal miners was confirmed, it seems to be advisable to consider the evidence obtained by these investigators as highly suggestive but not conclusive because great variations in the incidence rate of lung tumors occur among different groups of mice belonging to non-inbred strains.

However, some observations contradicting this conclusion have been reported by Lorenz, Heston, Eschenbrenner and Beringer as well as by Henshaw, Riley and Stapleton. Both groups of investigators found that mice exposed to ionizing whole body radiation revealed in addition to leukemia and ovarian tumors little, if any, increase in the number of pulmonary neoplasms. Of greater significance in this connection are the findings of Lisco and Finkel, who found in rats inhaling an aerosol of radioactive cerium, metaplastic and neoplastic proliferations of the bronchial epithelium. Similar results were

obtained with plutonium brought into the lungs of rats. Since uranium ore miners inhale not only radon and radium dust, but also uranium which may be retained in the lungs, Hueper, Zuefle, Link and Johnson injected metallic uranium powder dispersed in lanolin into the pleural and femoral cavities of rats and obtained sarcomas at the sites of injection in 13 or 24 percent of the 66 rats used. Evidence thus produced shows that focal accumulations of uranium which is an alpha-radiation emitter may exert a cancerigenic action upon the surrounding tissues, but it does not discriminate between the influence of metal toxicity per-se and radioactivity in the genesis of these lesions.

From a critical evaluation of the epidemiologic, clinical and experimental evidence available, it appears that a prolonged inhalation of radioactive gases and/or dust may elicit in man pulmonary cancers (Martland; Evans). In commenting on the production of lung cancer by atmospheric carcinogens, an Editorial (Lancet, 1952) remarked, "radioactivity of Joachimsthal mines is stated to be thirty times the tolerance dose. It is scarcely surprising, therefore, that in the past more than half the miners died of lung cancer." It stands to reason that this effect on the lungs of workers will prevail wherever similar conditions of exposure to radioactive gases and dust exist. The excessive suicide rate observed in the past among the miners in Joachimsthal (Sikl) reflects aptly the human misery produced if such hazardous working conditions are permitted to persist.

While there thus can be little, if any, doubt of the principal role of ionizing radiation in the production of lung cancers among radioactive ore miners and similarly exposed occupational groups, some comments on the possible significance of pneumoconiosis in eliciting or modifying this effect may be indicated.

Reports on the occurrence of pneumoconiosis among the miners in Schneeberg and Joachimsthal are contradictory. While Schmorl as well as Rostoski, Saupe

and Schmorl in their early reports (1936, 1928) noted that Schneeberg miners suffer from more or less intense anthracosilicosis and that this condition was causing or favoring the development of the bronchial cancers, Rostoski and Saupe stated in 1930 that pneumoconiosis was usually not very extensive in lungs with cancer. Because of the relatively slow course of the pulmonary tumors, they felt that pneumoconiosis may slow the intrapulmonary growth of the tumors. Hueck, on the other hand, remarked that silicosis does not represent a precancerous condition for the Schneeberg lung cancers. Some of the miners had silicosis but not lung cancer, while others had lung cancer but not silicosis.

Similar discrepancies seem to prevail concerning the Joachimsthal miners. Ziefl in 1935 reported that marked silicosis among these miners is quite frequent and that ashed lungs contain large amounts of silicon oxide. Pirchan and Sikle, on the other hand, maintained that no pneumoconiosis could be found in spite of an abundance of pneumatic drilling and that pneumoconiosis has no role in the production of the lung cancers. This opinion was shared by Löwy. Sikl in his most recent communication on the subject stated that some degree of fibrosis suggestive of silicotic origin could, of course, be seen in the lungs carrying cancer, and there were single cases of outspoken silicosis combined with cancer; but on the whole, silicosis was not a prominent feature in cases of cancer, on the other hand, the lungs most heavily affected with silicofibrosis being generally free from malignant growth. Behounek and Fort noted that pneumoconiosis was recorded as cause of death in only 8.2 percent of 63 miners who came to autopsy between 1929 and 1938. This statement contrasts strikingly with the observations made by Saupe during a chest x-ray study of 398 Joachimsthal miners conducted in 1939. He found that 43.4 percent of these miners presented roentgenologic evidence of pulmonary silicosis. However, silicosis was of minor degree among the seven (7) miners who were suspected of having pulmonary neoplasms.

Although the data are in part contradictory, it seems that silicosis does not play any significant role as a direct or contributory cause of cancer of the lung among the radioactive ore miners in Schneeberg and Joachimsthal. Whether it has an antagonistic effect upon the cancerization process or modifies the course of the established cancer remains problematical.

Finally it may be mentioned that these lung cancers vary a great deal in histologic structure, many were squamous cell carcinomas, others, round cell or anaplastic carcinomas, while a few were of adenocarcinomatous type. The radioactive lung cancers, thus, follow in this respect the pattern set by all other occupational cancers. The same environmental carcinogenic agent is capable of eliciting a great variety of cancers, although all of them originate from the identical tissue matrix.

Comments on Respiratory Radioactive Cancer

The rapidly growing production and use of radioactive material and thereby conditioned increased potentiality of inhalation of radioactive gases and dust for certain occupational groups and possibly, also, for parts of the general population determine the scope and significance of respiratory cancer hazards which may result from such exposures. Since the attack rate of lung cancer from this source is very high according to past experience with miners of radioactive ores, a competent assessment of the degree of exposure to radioactive dust, mist and gases by worker groups and neighborhood population is essential wherever radioactive material is produced or handled. The available information supports the view that the simultaneous inhalation of siliceous dust causing the development of a silicosis in individuals mining or processing radioactive ores does not represent a condition which favors the production of lung cancers or fundamentally modifies their course once they have become established.

E. Relation of Vegetable Dust Pneumoconioses to Cancer of the Respiratory Tract

An appreciable number of occupational activities of varied types are associated with an exposure to vegetable dust (tobacco, cotton, hemp, sisal, sugar cane, wood, paper). Some such contacts elicit pneumoconiotic changes. Inhalation of cotton dust given off in preparing the cotton for spinning and weaving causes a progressive fibrosis of the lung in reaction to the inhaled and retained cotton fibers (byssinosis) (Bolen; Thiry; Caminita, Baum, Neal and Schneider; Lanza). Pulmonary fibrosis, likewise, may follow upon a prolonged exposure of dust generated from the handling of sugar cane residue used for the production of fiber board (bagassosis) (Manas; Gerstl, Trager and Szczepaniak; Castleden and Hamilton-Paterson; Silson; Lanza). The possibility of a carcinogenic action of these vegetable fibers on the lung tissues has apparently not been considered nor have any cases of lung cancer among such workers been reported.

The inhalation of dust from dried tobacco leaves, on the other hand, has received some attention in this respect, although the actual existence of a fibrosing tobacco pneumoconiosis (tabacosis) is controversial (pro: Zenker; Boemke; Palitsch; contra: Long; Gross; Krueger, Rostoski and Saups; Pencoast; Miller and Landis; Landis, Funk, Smyth and Miller). In considering these claims, it should be noted that workers handling dried tobacco leaves not only have contact with the powdered vegetable matter of the tobacco leaf, but also inhale silica containing soil dust, nicotine, arsenical insecticide residues, and soot from the drying and curing procedure secondarily contaminating the leaves (McCormick, Smith and Marsh).

The claim that persons exposed to the inhalation of tobacco dust exhibit an excessive liability to lung cancer is partly based on epidemiologic evidence among German and Indonesian tobacco workers and cigarette manufacturers (Enger; Rottmann; Seyfarth; Brinkmann; Kouwenaar; Koelsch), in

part it is supported by observations made in connection with the high incidence of oral cancer among population groups indulging in the chewing of quids containing tobacco (betel quid-chewing tobacco (lit. Hueper; Wynder; Friedell and Rosenthal; etc.)). Although the evidence supporting a carcinogenic effect of inhaled tobacco dust upon the respiratory tract is at present only mildly suggestive, the strong evidence of such an action on the oral mucosa when tobacco is chewed, is an indication that serious efforts should be made to establish reliable factual evidence whether or not similar relations exist when tobacco dust is inhaled and acts upon lung tissue. Such investigations appear to be especially urgent, in view of the fact that recent experimental observations indicate that alkaloids as well as aliphatic polymers derived from cellulose possess carcinogenic properties.

F. Relation of Animal Dust Pneumoconioses and of Fibrosing

Pulmonary Parasitic Diseases to Cancer of the Respiratory Tract

Nothing is known whether workers engaged in the production, processing and cleaning of silk, wool and hair, and the various goods, made from these animal fibers, and thus, exposed to varying degrees to the inhalation of these proteinic macromolecular polymers have any excessive liability to cancer of the lung. While the occurrence of cancers of the skin among textile workers using such fibers has occasionally been reported, the causation of these lesions has justly been attributed to contact with carcinogenic lubricating oils.

In view of the repeatedly reported abnormally high lung cancer incidence among painters, it is of interest to mention briefly that the inhalation of shellac, a basic constituent of various lacquers, and oil and spirit varnishes extensively used in industry as finishing substances and often applied as a spray, has resulted in the development of a chronic indurative progressive fibroblastic pneumonia (Hirsch and Russell). Shellac is a natural

resin of insect origin and before purification contains lac dye and wax. Most of these impurities except wax are removed in processing the shellac. Some evidence favors the view that the component resins of the original lac are bound together in a complex form or a weak chemical linkage (Bhattacharys). This relationship is destroyed during purification and the pure lac resin consists of hydroxy acids of the aromatic and aliphatic series joined together by lactone and various internal ester linkings.

Although printers and cabinet makers are exposed during their occupational activities to many other chemicals, some of known carcinogenic nature (solvents oils, resins, plastics, pigments, waxes, paint removers), it may be wise to include shellac in any future considerations of the possible causes of the excessive liability of members of this occupational group to lung cancer.

Among the various parasitic infections of the lung which are associated with localized or diffuse fibrosing changes in this organ, tuberculosis stands in first place. In fact, during past decades, pulmonary tuberculosis, particularly tuberculous cavities and bronchiectases, were regarded by some investigators as a frequent cause of cancer of the lung (Ewing; Kraft; Fried; Derischanoff; Larson; Moore and Neal; Moore and Schmeisser; Ssipowsky; and others). The coexistence and possible causal, antagonistic or fortuitous relationship of tuberculosis and cancer of the lung has been the subject of numerous investigations (Hueper; Marx; Cohen; Munteau and Amon; Schwartz; Robbins and Silverman; Hambly). The possibility of a causal relationship was suggested because cancer seems to develop with unusual frequency in tuberculous lesions of the skin (lupus) (lit. Hueper). The great majority of investigations, however, does not support the contention that the various chronic inflammatory and fibrosing processes associated with pulmonary tuberculosis causes or favors the development of cancer of the lung (Fried; Brymalski and Sweany; Attinger; Olson; Hambly; Vinson; Symmers; Walzer; and

others (see lit. Hueper)) and indicates that the coexistence of these two conditions is fortuitous. This conclusion is supported by the fact that cancer of the lung has become in recent years much more frequent, while pulmonary tuberculosis, during the same period, has shown an opposite trend. Cancer of the lung, on the other hand, is of great importance for differential diagnostic reasons, since the number of cases in which tuberculosis and cancer of the lung coexist appears to be increasing (Gerstl, Warring and Howlett). Cancer of the lung may symptomatically simulate pulmonary tuberculosis (Siltzbach; Bergmann, Shatz and Flance; Hembly).

The causal relationship between schistosomiasis of the bladder and cancer of this organ is a generally acknowledged fact. Less certain is an etiologic connection between cancer of the colon and rectum and schistosomiasis of these organs. Although schistosomiasis of the lung is not infrequently observed in regions in which this parasitic disease is endemic (Egypt; North, Central and South Africa; Central and South America; West, East and South Asia) (Erfan; Gelfand; Mainzer; Shaw and Ghareeb; Cawston; Wright, et al.; Clark and Graef; Koppisch), the ultimately developing pulmonary cirrhosis apparently has not given rise to cancer of the lung as far as the published records indicate.

The available evidence, thus, apparently shows that the inhalation of animal fibers or the presence of parasites and their metabolic products in the lung does not result or favor the development of cancer in this organ. It cannot be maintained, however, that the quantity and quality of data existent on this subject should make this conclusion definite.

G. Relation of Various Bacterially or Chemically Induced Chronic

Inflammatory Processes to Cancer of the Lung

Although many noxious gases, vapors and fumes do not accumulate in the lungs on prolonged exposure, they may elicit chronic inflammatory processes

of more or less diffuse or localized character (chronic bronchitis, bronchiectases, chronic chemical pneumonia, cavitation). Occasionally, such exposures and their anatomic effects upon parts of the respiratory tract have causally been related to cancers subsequently developing in the injured and inflamed tissues.

Birkholz asserted that a laryngeal carcinoma was caused by an inhalation of benzine-petroleum vapors over a period of three (3) years. Commenting on a similar claim of Nikoleff that the inhalation of benzene vapors was responsible for the production of a pulmonary carcinoma, Koelsch stated that benzene and its homologues do not exert a specific carcinogenic effect upon the lung tissues. It is, however, uncertain whether or not this statement applies without any exceptions to all organic solvents and solvent vehicles, since some of these agents have elicited cancers when applied to the skin of mice. Although repeated exposures to formaldehyde fumes, such as those sustained by manufacturers of certain plastics, embalmers, makers of certain deodorizers, may cause chronic bronchitis (Wyss) and despite the fact that formaldehyde has recently been shown to be a mutagen (Auerbach), there does not exist any valid evidence indicating that this chemical also is a respiratory carcinogen.

Claims have repeatedly been advanced concerning carcinogenic effects produced by the inhalation of alkali and acid fumes upon various parts of the respiratory tract (larynx, lung) (Kikuth; Hunemann; Bethe). A highly doubtful significance of such isolated occurrences is indicated by the fact that Koelsch did not find a single case of lung cancer when studying large series of manufacturers of inorganic acids (sulfuric, hydrochloric, nitric acid) and large scale industrial users of these chemicals (chemical, metallurgic, and graphic industries). Likewise, there is no evidence that the pneumo-fibrosis observed in a high percentage of Italian sulfur miners causing

peribronchial as well as micronodular fibrotic lesions associated with bronchiectases increases the liability to cancer of the lung.

Following the first World War the alleged carcinogenic action of war gas poisonings has played a conspicuous role among speculations as to the cause of the rising number of respiratory cancers. Poisonings by chlorine, phosgene, diphosgene, dichlorethyl sulfide, and diglycol chloride was considered one of the major causes in this development by Kikuth; Spaner; Brockbank; Matz; Denker; and others. Koelsch conceded that a few cases of lung carcinoma exhibited a doubtful etiologic relation to war gas injuries. Although the chronic effects of acute war gas poisonings were obliterative bronchiolitis and bronchiolar metaplasia, a connection between cancer of the lung and war gassing was lacking in the series of pulmonary cancers reported by Berblinger; Probst; and Simons. Since some of the war gases contained arsenic, while derivatives of mustard gas have recently been shown to possess carcinogenic properties for experimental animals, a final decision on the existence of such connections must at present be held in abeyance.

The relative infrequency with which other chronic inflammatory and cicatricial pulmonary lesions, especially bronchiectases, of nonspecific, and in part traumatic nature, appear as alleged direct or indirect causes of bronchogenic carcinomas (Schwyter; Friedrich; Rössle; Mangelsdorff; Woodruff and Nahas; Weller; Plazy; Gouriou and Germain; Dahlmann; Wells and Cannon; Pilgersdorfer; Luckow; Gomez; Fischer-Wasels; Fischer and Fenster; Gillespie; Bourret and Fraisse; Rospide; Konwaler and Reingold; and others), indicates that chronic irritation (Berenblum) as such does not represent a direct and primary cause of cancers of the respiratory tract. The validity of this general conclusion is apparent from the total evidence presented.

It may be concluded that pneumoconioses of benign or progressive type as well as pneumofibroses resulting from the inhalation of noxious gases,

vapors and fumes not retained in the lungs or from bacterial or parasitic pulmonary infections of occupational or non-occupational origin do not exhibit a consistent causal relationship to cancer of the lung.

Wherever cancer of the lung appears to be etiologically connected with the inhalation of chemical dusts, fumes, mists, vapors and gases, such relations are highly selective and specific. Since pulmonary cancers are also elicited by chemical agents which do not produce any appreciable fibrosing reactions in the lung, it is most unlikely that these pulmonary changes represent an essential primary part in the causative mechanism of pulmonary neoplasia. It is quite evident that the irritative and carcinogenic properties of respiratory carcinogens are neither identical nor closely interrelated, and that in this respect, these agents behave like those which cause cancer of the skin. Only a few of the skin irritants are also cutaneous carcinogens.

While observations in other fields of human and experimental carcinogenesis indicate that acute or chronic inflammatory processes might set off or accelerate a cancerization process initiated by a specific agent, there is no evidence available indicating that such an action is exerted by pneumoconiotic reactions upon the development of lung cancer. The average latent periods of occupational respiratory cancers is about the same for cancers elicited by all respiratory carcinogens investigated, regardless of whether they have pneumoconiotic qualities or not.

Chemoallergic reactions, however, may be active in the production of both fibrosis and/or cancer by some of these agents. The evidence on hand does not permit a definite conclusion whether the action of a pneumoconiotic agent or the presence of a pneumofibrosis accentuates or impairs the action of a respiratory carcinogen or has no influence whatsoever on it. It is, likewise, uncertain whether a progressive pneumoconiosis favors or impedes

the intrapulmonary and metastatic spread of an established environmental or cryptogenetic cancer of the lung, although the general evidence available on this point would support a hindering effect.

H. Methodologic and Medicolegal Aspects

It has recently been asserted that air pollution is not a serious or critical menace to public health (Ianza). The evidence presented does not support the correctness of such a view. The industrial hygienist accustomed to think in terms of toxic concentrations of chemicals in the atmosphere should become better acquainted with the often subtoxic or metatoxic concentrations in which carcinogens can and do act effectively. The possibility of delayed untoward effects by the prolonged inhalation of atmospheric pollutants appears to be especially likely, whenever such pollutants exert a cumulative effect or are readily retained in the body and, thereby, able to perform their insidious work over long periods of time. The long, symptom free latent periods sometimes extending from 15 to 20 years after cessation of exposure to an environmental carcinogenic agent are striking and convincing proof and demonstrations of this fact. This consideration applies not only to industrial products and wastes, but with equal force also to the large number of environmental poisons (pesticides, herbicides, chemical additives to foodstuffs, cosmetics, and sanitary household goods) with which large parts of the general population come in daily contact. The recent demonstration of cumulative storage of DDT, which when given in excessive amounts to rats, has produced hepatic tumors, in the fat tissue of unexposed members of the general population, provides an apt illustration of the possible existence of health hazards from such sources (Pearce, Kattson and Hayes).

While it may be difficult in many instances to supply absolute scientific proof, which will satisfy even the biased sceptic, of relationship between air contaminants and damage to health (Best), particularly cancer of the

respiratory organs, such proof can be obtained if competent personnel, adequate funds and facilities are made available. Past efforts made in this respect were scarcely extensive nor always competent.

Environmental carcinogens behave very much like pathogenic microorganisms in producing disease phenomena in only a fraction of the total population at risk. As a rule, the number of persons affected depends directly on the relative potency of the carcinogenic agent and on the intensity and duration of exposure to it by the individuals composing the group. While naturally susceptible individuals seem to succumb first and in the presence of a weakly acting carcinogen may be the only persons affected, the attack rate may practically reach 100 percent of the exposed population, if a carcinogen is highly potent and the exposure to it very severe and prolonged.

Consideration, also, must be given to the fact that in contrast to the almost simultaneous affliction of large numbers of persons by infectious diseases during epidemics, environmental and occupational cancers exhibit in this epidemiology a distinctly endemic pattern, i.e. the existence of an environmental or occupational cancer hazard becomes apparent only if effectively exposed and, therefore, restricted population groups are analyzed for the occurrence, number, site, age and sex distribution of cancers observed among its members over periods of not less than five (5) to 10 years. The inclusion of a large number of unexposed or little exposed individuals who did not exhibit at any time the specific stigmata of an effective contact, invariably leads to a dilution and possible obliteration of the actual positive evidence (Hueper; Hueper and Mancuso). Whenever occupational cancers are consistently associated with other disease phenomena affecting the same organ and producing symptomatically similar lesions such as cancer of the lung and pneumoconiosis, reliable epidemiologic evidence depends upon the availability of pathologic diagnoses (biopsies, autopsies) for all cases included in the group evaluated.

Chest x-ray studies and cytologic data from sputum examination are under such circumstances of only limited value.

It is obvious that an efficient and reliable study of respiratory cancer hazards occurring in industry is a complex undertaking requiring not only competent investigators and adequate facilities but also a comparatively unusual amount of financial support. A proper appreciation of the actuality and scope of the problem among interested parties is of rather recent date and so far only a few research institutions have devoted a part of their work to a study of industrial respiratory cancer hazards. The Trudeau Laboratories have pioneered in this field and occupy a leading place. Because of the rapidly increasing importance of respiratory cancers as public health problems, the National Cancer Institute has provided through special cancer control grants, financial assistance to three (3) universities (University of Pittsburgh, University of Utah and University of Southern California) for the establishment of research facilities for epidemiologic and experimental studies of environmental cancer, particularly cancers of the respiratory tract caused by occupational and environmental air pollutants. It is hoped that these units may become within a few years, organizations totally independent from governmental support and may provide interested private parties as well as local health agencies with additional privately controlled special laboratories, staffed by cancer research specialists competent and experienced in highly diverse and difficult aspects of environmental cancer research. It may be hoped that with the passing of time, an effective and reasonable control of environmental respiratory cancer hazards may be achieved, which may prove satisfactory and fair to all parties concerned.

were carcinogenic in the lung of man. I do not know of the evidence that pure benzpyrene, per se, will produce cancer in the primate. Does such evidence exist?

BY DOCTOR HUEPER:

No, that evidence does not exist. There is only some circumstantial evidence available. There are two cases on record among laboratory workers which, by accident, came in contact with benzpyrene. One was the screen of the skin while later on a cancer developed, but whether that is conclusive evidence, I would - I am not quite certain, but we have to consider one fact, that benzpyrene is only one of the carcinogenic chemicals in these materials. For instance, Falk and Steiner isolated three or four, and they are all shale oils available which do not contain the pyrene, but which are carcinogenic, so we should not - it isn't the material as such, the crude material as such, which we should consider as carcinogenic, and we should not take the presence of one carcinogen as authentic, but should take into account that such materials are carcinogenic.

BY DOCTOR RHOADS:

This was one of the things I hoped the doctor would bring out. If benzpyrene is present, it suggests the presence of possibly stronger carcinogenic agents, or a whole series.

BY DOCTOR HUEPER:

Discussion

DR. RHOADS: Dr. Hueper, you referred several times to the presence of benzpyrene in suspected material as supporting the view that these materials were carcinogenic in the human lung. Does evidence exist that pure benzpyrene, per se, will produce cancer in man?

DR. HUEPER: There is no direct evidence, only circumstantial evidence. There are on record two cases among laboratory workers who accidentally came in contact with benzpyrene. One was of the skin later a cancer developed but whether that is conclusive evidence I am not certain. We must remember that benzpyrene is only one of the carcinogenic chemicals in these materials. Falk and Steiner isolated three or four. There are shale oils which do not contain benzpyrene but which are carcinogenic, so we should not consider only the crude material, as such, but should take into account components that are carcinogenic.

DR. RHOADS: The presence of benzpyrene suggests that possibly stronger darcinogenic agents, or even a whole series, are alos present.

DR. HUEPER: A whole spectrum of carcinogens.

DR. SAMUELSON: With reference to carbon black I would like to correct the record. The Steiner work showed that 3:4 benzpyrene and 1:2 benzpyrene, a very mild carcinogen, was present in the extract of carbon black, and no evidence of carcinogen to follow.

DR. RHOADS: Dr. Hueper's joking remark that the sun might be prophylactic raises an interesting point. I believe that one cannot wholly ignore the endocrine system in cancer, in view of the surveys of McCann, Tannenbaum and others. Other surveys not yet published suggest very strongly that hypophisectomy will render inactive certain compounds which otherwise are active and exceedingly carcinogenic and it is not inconceivable that the sun might play a similar role.

DR. HUEPER: Pellet, who studied the incidence of cancer of internal organs among Canadians, suggests that persons who develop cancer of the skin become partially immune to cancer of the internal organs. I do not believe that the evidence, from an epidemiological viewpoint, shows that. If an individual is exposed to an occupational agent that causes cancer of the skin, he has, in fact, a greater liability to develop cancer of the internal organs. A pathologist of the University of Richmond has made claims similar to those of Pellet but I do not believe that they are justified on the basis of the factual evidence.

DR. RHOADS: Dr. Levin, will you comment on the statistical validity of the evidence regarding occupations for which the incidence is comparatively low? Many of those figures are from the Kenilway series; do you wish to comment on the validity of those observations of the engineers, the tobaccoists, the barroom workers, and individuals having other occupations?

DR. LEVIN: I haven't examined the data sufficiently to be at all critical. It is interesting that with a few exceptions, such as the lung cancers of Schneeberg, the majority of persons exposed, even to a high dosage, do not develop a disease. This condition suggests that if we confine our attention entirely to ventilation and ignore the intrinsic constitutional agent we are over-stressing the situation. We should attempt to determine why some persons exposed to a specific concentration of a carcinogenic agent do not develop disease while other persons do.

Some of the published material to which Dr. Rhoads referred is difficult to evaluate because factual information is lacking: How many persons were in the factory at a certain age for how many years; were all the cases studied; at what age did they retire; was there a difference between the people you did have information about and the people you did not have information about? As a state official I would say that the evidence is sufficiently strong to justify the proposal that industry be required to keep, confidentially and without bias, much better records than it does regarding both its workers and what happens to them. The information is there; we simply are not getting it in such a way that we can analyze it.

DR. RHOADS: I should like to emphasize Dr. Levin's point that it is perhaps the obligation of industry to set up so-called preventive experiments with

control groups which will provide possibly the most final data.

DR. PRATT: With referenced to carcinoma in individuals with asbestosis, we see hyperplasia of the bronchiolar epithelium and extension into the alveolar spaces in areas of the lung that show fibrosis, but if we look at the epithelium of the major bronchi we see a normal appearance. Nevertheless, the cases that have carcinoma with asbestosis - at least the cases I have seen - in the main give every evidence of being primary in the major bronchi. It seems to me that if there is a connection between asbestosis and cancer, it must be a considerably more devious one than simply hyperplasia, metaplasia, and neoplasia. I would like to hear Dr. Hueper's comments on this point.

DR. HUEPER: I am glad you mentioned this topic because it is of great interest to me. The carcinogens are really misnomers. If you study the anatomic reaction to carcinogens, you see first hyperplasia; you see many benign tumors and then, finally, you may see carcinomas - some of the benign tumors may have become malignant; or you may see, in the prepared soil, carcinomas of primary origin. Personally, I place a great deal of significance upon the development or history of carcinomas in the histologic sections of the cases I have studied. I believe that those sections give us the life history of the carcinogen and the life history of the cancer. To me, the changes of asbestosis are a typical example. I have seen characteristic changes in the cancers we produce in dogs, in the cancers we produce in mice with the various hydro-carbons. Those progressive changes are always significant.

DR. DRINKER: As I was the instigator of two of the surveys Dr. Hueper mentioned - Lombardo's on arsenic and Ingall's on carbon black - I have more than a casual

interest in Dr. Hueper's remarks. Today the big arsenic producer is Sweden, with Mexico second and the United States third. We shall continue to produce arsenic for an indefinite period because copper ores, which are smelted, contain arsenic. The industrial experience of the smelters in the United States dates back for about 40 to 50 years - a relatively long experience for health matters; the Mexican and the Swedish experience is briefer. The survey on arsenic was instigated because we were a little apprehensive, after finding that chromate workers get perforated septa and lung cancer, that perhaps the same thing might be caused by exposure to arsenic.

Dr. Hueper questioned the interpretation of Lombard and Snegireff's statistics, in which they concluded that the handling of arsenic trioxide does not produce a significant change in cancer mortality. The incidence of 50 per cent, to which Dr. Hueper refers, is quite startling but the numbers of workers involved were so small that the high rate could have been merely a matter of chance. Lombard and Snegireff used for a control a smelter that handled no arsenic yet the incidence of lung cancer of the control was a little greater than that of the community as a whole. In Utah, Neal and others made a good survey concerning arsenic; they found no evidence of lung cancer.

I believe that Dr. Levin's plea for better statistics in industry is an extremely good one. I know that the big smelters are anxious to keep good records.

DR. RHOADS: There is a tendency to incriminate pure compounds of known activity when considering the possible carcinogenic action of a complex mass of materials coming out of a smelter or out of a complex organic mixture. The evidence may be good for the problem involved - contamination or pollution - but not so good for a single pure constituent of the polluted material.

DR. HAMPE: Dr. Pratt mentioned that in carcinoma with asbestosis he did not find the so-called hyperplasia, metaplasia, and so on, of the carcinoma. In Holland I have had two cases of asbestosis, one with carcinoma and one without. I tried to differentiate between the type of hyperplasia seen in tuberculosis, so-called metaplasia, and the type ordinarily seen in bronchial carcinoma.

marks
DR. PRATT:

A whole spectre of carcinogens.

BY DOCTOR RHOADS:

Doctor Hueper made also a very interesting point in joke, that was the disposition of the point that the sun might be prophylactic. I think one can not wholly ignore the endocrine system in cancer, in view of McCann, Tannenbaum's and other worker's surveys. There are surveys as yet unpublished, but they suggest very strongly indeed that hypophysectomy (?) will render inactive, inert compounds which, otherwise, are active, exceedingly carcinogenic, and it's not entirely inconceivable that the sun might play some role.

BY DOCTOR HUEPER:

Various surveys have been made by Pellet, who studied the incidence of cancer of internal organs among Canadian - among the natives, and he suggests that persons who develop cancer of the skin would become partially immune to cancer of the internal organs. I don't think the occupational evidence shows that. If you are exposed to an occupational agent that causes cancer of the skin, you have, in fact, a higher liability to develop cancer of the internal organs. That's the general evidence offered from a general epidemiological viewpoint.

The pathologists of the University of Richmond - his name escapes me right now, he makes very similar claims.

I don't think they are justified on the basis of the factual evidence we have. One can speculate a lot but I think it's our feeling we have to stick quite strictly to what we actually observe, the factual evidence, and not speculate too much.

BY DOCTOR RHOADS:

Now, I'd like to ask Doctor Levin to comment on the statistical validity of the evidence regarding occupation, where the incidence is comparatively low. Many of those figures are from Kenilway series, of course; do you want to comment on the validity of those observations of the engineers, the tobaccionists, the barroom workers, other happy occupations?

BY DOCTOR LEVIN:

I don't think I have recently examined that data to - sufficiently to be at all critical. One of the things which impressed me about the type of information that Doctor Hueper presented and which is, I think, good literature is that, in general, even at the - with a few exceptions, the lung cancers of Schnaberg, seems to be one of those, even with the high dosage of exposure and the majority of people exposed, apparently do not develop a disease, which strongly suggests that we are over-stressing the situation if we confine attention entirely to the ventilation and ignore the intrinsic constitutional agent. I think that perhaps - I

don't mean to say that we should do the other thing and throw overboard the evidence regarding these things, but I think we should study just as carefully why it is that some people just as exposed to the concentration of the agent, do not develop the disease. I think we may get clues to the development of the disease in others which may be more useful or certainly as useful as the preventive aspects of the occupational data.

Some of the published material that Doctor Rhoads referred to is simply of a kind that's very difficult to evaluate because you don't have enough facts. You don't know how many people were in the factory at a certain age for so many years. You don't know whether all the cases were studied. You don't know at what age they were retired. You don't know the difference between the people that you did have information about, and the people that you did not have information about, but from the standpoint of a state official, which I am, and from the standpoint of what all this sort of thing leads to, from the standpoint or question of actual action, official action, I would say - and since this is an industrial group, I'd like very much your reaction to this proposal - I would say that the evidence is sufficiently strong to at least justify one proposal and that is that industry be required to keep much better records than it does regarding both its workers and what

happens to them without any bias as to what conclusions will be reached and perhaps with every preservation of the confidentiality of that information.

I think the information is there that will answer many of these questions beyond a shadow of a doubt; we simply are not getting the information in such a way so that we can analyze it and really be sure of some of these things. I think, however, that could very readily be done.

BY DOCTOR RHOADS:

Thank you, Doctor Levin. I hope that point will come out and I think I would like to emphasize another point Doctor Levin made previously, and that is it is perhaps the obligation of industry to consider the setting up of the so-called preventive experiments with control groups, which, after all, will provide perhaps the most final data, I take it, from your feelings about it.

Like to have your further discussion. Yes, sir.

BY DOCTOR PRATT:

In reference to carcinoma in asbestosis cases, we see the hyperplasia of the bronchial epithelia and its extension into the alveolar spaces in areas of the lung that show fibrosis, but if we look at the epidemiology of bronchi, it seems to me it looks quite normal; yet in the cases that do have carcinoma together with their asbestosis, the cases that I've seen, in the main give every evidence of

being primary in the major bronchil. It seems to me that if there is a connection between asbestosis and cancer, it must be considerably more devious than simply hyperplasia, metaplasia and neoplasia. I'd like very much to hear Doctor Hueper's comments on that point.

BY DOCTOR HUEPER:

I'm glad you brought up that question, because it's been a long time in my heart, but I'm glad you brought up that question because it's been a long time on my heart, and I would like to shoot it off.

The carcinogens are really misnomers. If you study the ^{in vitro} - an atomic reaction of carcinogens, you see first hyperplasia, you see lots of benign tumors and then finally you may see carcinomas, you may see that some of the benign tumors become malignant or you may see, in the prepared soil carcinomas, originating primarily.

I personally place a great deal of significance upon seeing the development or history of carcinomas in the slides I have studied in the cases I have studied. I think they give us the life history of the carcinogen and the life history of the cancer. To me, the changes in asbestosis are typical of that. I have seen them in the cancers which we produce in dogs. I have seen them in cancers which we produce in mice with the various hydrocarbons; it's always the same story; to me those progressive changes are

significant.

BY DOCTOR RHOADS:

I think this is a very interesting point that comes up frequently in discussions of the histo-pathologic development of neoplastic change, and always puzzled by one group of cells presumably equally in contact with others become exposed to a rapid shift in mortality, and I presume there are unknown factors regarding concentration. Next question?

BY DOCTOR DRINKER:

I was the instigator of two of the surveys Doctor Hueper mentioned, the one of Lombardo's on arsenic and the one of Ingall's on carbon black, so I have an interest, more than a casual one, in Doctor Hueper's remarks. Today the big arsenic producer is Sweden, with Mexico second and the United States third. We will continue to produce arsenic for the indefinite future, because of the fact that the copper comes contaminated by arsenic.

The industrial experience of the smelters dates back now in the United States for about forty to fifty years, so that as experience goes in health matters, they probably have as good as there is. The Mexican experience is a little briefer and so is the Swedish experience. The survey in the arsenic works was instigated because we were a little apprehensive after what was found in the

chromate industry to which we may have some slight analogy.

That is, the chromate workers get the perforated scea, and then they were shown to get lung cancers, so we wondered if perhaps the same kind of thing did not occur in the arsenic business.

Doctor Hueper remarked that Lombard, and Lombard and ^{Snegireff} Schnager, from an interpretation of their statistics, the interpretation was curious. I can't say I agree. I will admit that half of something is - startles you, and - but half of four or half of ten is not very startling, and I rather think that the explanation in the smelter in the west in which Hueper takes them to task for not making more of the arsenical cancer is due to the fact that the figures are too low, the amount of numbers of workers are too low and they concluded that it might just as well have been the other way, on a pure chance basis.

They took for a control a smelter in the arsenic business that had no arsenical exposure and, in that case, the lung cancer, I regret to state, was a little more alarming than that of the community at large, and there was no arsenic exposure whatever. The smelter handled no arsenic.

I think that the doctor's plea for better statistics in industry is an extremely good one and I know that the smelting business, all of the big smelters now, I'm familiar with what they're doing about this, are more than

anxious to get their figures straight and will keep records. Their records, in the past, are very fragmentary. Hueper did not mention the fact that there was a good arsenical survey in Utah by Neal and others, in which they found no evidence of lung cancer.

BY DOCTOR RHOADS:

Well, here information comes from another angle, perhaps alluding to a point brought up before, and that is the tendency is to incriminate pure compounds of known activity when one is considering the possible carcinogenic action of a most confusing and complex mess of materials coming out of a smelter or coming out of a complex organic mixture, and perhaps the evidence, I take it the evidence is good for the problem involved, in contamination and pollution, but not so good for a single pure constituent of the polluted material. Yes, sir?

BY DOCTOR SAMUELSON:

On that point, in reference to carbon black, I'd like to correct the record. The Steiner work showed that 3:4 benzpyrene and 1:2 benzpyrene, a very mild carcinogen was present in the extract of carbon black, and no evidence of carcinogen to follow.

BY DOCTOR RHOADS:

Thank you. Will you give your name please?

BY DOCTOR SAMUELSON:

Doctor Samuelson.

BY DOCTOR RHOADS:

Very interesting point, yes.

BY DOCTOR HEMPE:

I would follow up the question of Doctor Pratt. Did I hear it right that you were commenting on the fact that in asbestos carcinoma or carcinoma with asbestosis, you did not find the so-called hyperplasia, metaplasia and so on, ^{located in bronchial} of the carcinoma? Was that what you said?

BY DOCTOR PRATT:

That's what I said.

BY DOCTOR HEMPE:

May I show one slide? I come from Holland and there were only two cases up to now, of asbestos in Holland, one with carcinoma, one without. It was by chance I got them; it was a chance I got the case with hyperplasia and metaplasia. I don't know how you use these words, hyperplasia, metaplasia, I can only say that I tried to differentiate between the kind of hyperplasia, as you see it in tuberculosis. I tried to differentiate between the types, as you see it in tuberculosis, so-called metaplasia and the type you see ordinarily in carcinoma, bronchial carcinoma of the lung, as you can study it in specimens, and then I must say that in the same type of bronchi, the middle lot, not only the small ones, but the greatest ones,

as you will get the specimens, but in the middle grades bronchi, you will find a certain type of metaplasia, that certainly is related to carcinoma.

I do not say that carcinoma does come from a type of metaplasia, but you do find them together, as you do not find the metaplasia in bronchiectasis or tuberculosis, and this is the slide I got here. Our slides are not as beautiful and our micrographs are not as beautiful as you normally see them here, but I hope you will see the point in what I am trying to show.

Here you see the bronchi epithelia, and you see - the comment will come that is not an entire cross-section, but you will see the typical metaplasia, the hyperchromatosis of it, and you will see entirely how this bronchial epithelium is out of order.

This is one of the greater bronchi; it is not a small one, and the next please. Here you see the contents of the middle lobe bronchus, mucous cells, mucocytes. Here you must imagine the basal membrane and here you see the so-called typical metaplasia, perhaps leading to hyperplasia, I don't know.

These are of the greater bronchi too, and the next please? Here you see the same, that thick mucous that is coughed up so difficultly in asbestosis, the thickened basal membrane, and here the very typical kind of epithelium

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DR. RHOADS: You say that the mass survey did not reveal these cases but that the roentgenograms taken when the individuals applied for medical care did?

DR. CARTIER: No, the roentgenogram was taken during a routine examination at the clinic.

DR. RHOADS: How does that differ from the mass survey?

DR. CARTIER: It differs in that people over 50 years of age do not take advantage of the mass survey. All the cases have been detected at the clinic and, later, have been followed up and have come to autopsy.

DR. RHOADS: It appears that somehow the mass survey doesn't always result in the actual detection of cancer and in the bringing of the patient into medical hands.

that certainly is quite an interesting thing in this case.

The next? There you have the same in the peripheral parts. I don't know if it were alveolar or not, and there you have the same as Doctor Hueper showed, that same metaplasia or, in any case, a typical form, let us call it epithelium, and diffusing - diffuse, that is going into carcinoma.

This is a case where I could not prove that carcinoma really was due to asbestos, and I think no one up to now proved it, although we saw many cases of them. It was a case where carcinoma of the lung was multiple and I think that follows up the question of Doctor Pratt too, when he commented on the fact that the metaplasia was more extended in the peripheral parts of the lung than you would expect that you would find more carcinomas in asbestos lungs, and I had one of these cases only I could not prove that it was bronchial carcinoma, so one of the questions of Doctor Pratt is fulfilled here.

Here you see the peripheral metaplasia with carcinoma, but in the periphery, but together with it you see the metaplasia in the bronchi, the greater bronchi.

BY DOCTOR RHOADS:

Thank you very much, thank you, Doctor Hempe. To keep on schedule at all, we are sort of late, we'd best now have a break until twenty-five minutes of twelve.

(Short recess taken)

BY DOCTOR RHOADS:

The next paper on the Symposium concerns the paper already discussed by Doctor Hueper. If we could have a little quiet, it would be very helpful for those who are presenting papers. Doctor Hueper has asked to comment on Doctor Drinker's remarks. I think we must defer this until we can get the papers under way.

I will, therefore, introduce the paper on asbestos mining by Doctor Cartier. Doctor Cartier.

BY DOCTOR CARTIER:

Members of this Symposium: Not being a statistician, neither an epidemiologist, I don't think I should have accepted to present this paper, but I think, to excuse myself, I should say that this statistics and most of the papers I am presenting have been reviewed by your statistician in Montreal and Doctor Phillips, official statistician for National Cancer Institute of Canada in Toronto, so I would like that these two experts take the responsibility of the statements I might advance.

So I was invited to present this paper on the pulmonary cancer in the asbestos mining industry, not on account of any special qualification, only because I happen to be in charge of a clinic which has, as a primary function, to supervise the primary condition of four thousand asbestos miners, such a group of entrants offering an

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So I was invited to present this paper on the pulmonary cancer in the asbestos mining industry, not on account of any special qualification, only because I happen to be in charge of a clinic which has, as a primary function, to supervise the primary condition of four thousand asbestos miners, such a group of entrants offering an

outstanding metallurgical study of cancer of the lung in relation to asbestos fibers or any other related fibers.

In the origin of mines, six different companies are doing asbestos mining operations, so for more than eighty years, and since 1945, these companies have organized the Thetford Industrial Clinic which has, as a primary aim, to fulfill a program of industrial hygiene, mainly medical and with dust control provisions.

In 1950, the concept of an inhalation of asbestos fibers and pulmonary cancer was mentioned by many investigators and it was at that moment that the Medical Advisor or the Quebec Asbestos Mining Association, saw advantages for the employees and the employers, to recommend a company investigation composed of an epidemiological and experimental study.

This report is part of this study.

The Thetford Industrial Clinic has been in operation only since 1946, and we have medical records on five thousand fifty-two employees, but this survey is based only on three thousand nine hundred fifty-seven workers having worked in the industry from two to sixty years. About one thousand employees have been rejected from this survey because they have not worked long enough, or because they have worked in different industrial trades before.

Before presenting the first table, I think it is

worthwhile to say that the labor turn-over in the asbestos industry is one of the lowest in all the industry in Canada, meaning that the stable population is working in our mine and meaning, also, that in many aspects, it is easier to study and more representative.

The first slide please. So as I mentioned, this Table Number 1 shows the age group distribution of the group study and I don't think it needs any comments except to say that the average age of our employees is thirty-five, about two years older than the average for the general industrial population, and this table will show also that 1,577 men are included in the age group from forty to - and over, in the group where the incidence of cancer is more general.

Second -- next table, please. This table indicates the number of years spent in the asbestos mining industry by the total number of employees surveyed. Of course, this table is not as significant as would be a table indicating the specific exposure of these employees, but after many attempts it has seemed impossible to - to add together the different years spent in different concentration of fibers, to which most of the employees have been exposed, and I think as a remark too, it is worthwhile to mention that two thousand, two hundred have spent more than ten years in the asbestos industry.

Next table, please. The following table shows a classification of various exposures in the asbestos mining industry. Realize that this classification could be discussed for many hours, and present many quarrels, but I think nevertheless that we need, for many purposes, we need a job classification, even for the exposure aspect or for research, so after we were stationed with our engineer, we took the average of all the dust survey made in the mining industry and we decided just to draw this table, and this table has also, I don't know if it is objective advantage, but has the advantage of correlating with our case of asbestosis.

I want to state that all the cases of asbestosis have belonged - have worked in shops, under that rule, I'm sorry, in the lower group, and our men having worked even from two to forty-five years in that kind of exposure, has never developed an asbestosis which was found by pathologists or epidemiologists or by experiments, to be of any sequence, any medical sequence.

This table, as you may see, presents million particles per cubic foot and the percentage of fiber count. This is very important, because from experience to experience, it seems that the fiber is the only pathological agent, rock dust not being - not being reproduced in any consensus, pulmonary fibrosis. I think, only this might

need some further explanation, because you see that is not clear. Even this might seem a little less concentrated, but just the same we put in this whole group, because on the ground, dry work and the grizzly men are working longer, they are working in more constant concentration of fibers, and in the third place, because we have found a few exceptional cases of asbestosis in that group. So, please remember the categories, 0, 1, 2, 3, 4 and 5, because in the coming tables, they might help you to understand them.

The following tables intend to indicate incidence or prevalence of pulmonary cancer in different groups of asbestos workers in comparison to different control groups. The task of selecting significant control group has been, in this case, as everywhere, very difficult. We would have wished to be able to find a group in the province of Quebec, a group in any other comparative - comparable industrial group, but it was impossible for the following reason, that in no other industrial cities in Province Quebec, so large a proportion of the adult population is followed medically so closely, because I think I should mention to you that about thirty-three - about thirty percent of the adult population in Thetford are working in the mines, being the only industry, so we can't have a very close supervision of the function of the - of the condition of the lungs.

We tried, with the mass survey, to detect a com-

parable survey group, but I don't think it was or is possible, because in the Province of Quebec, very few adults over fifty years of age are taking advantage of the mass survey. As a matter of fact, the mass survey made in Thetford mines in 1950 by the Provincial Department of Health, did not detect a single case of pulmonary cancer, either in the generic operation or in fourteen miners who have been X-rayed, so having not succeeded in selecting a good control group for the reason just mentioned, we have decided to make comparative study of incidence of the pulmonary cancer in different groups of asbestos workers, which can be studied with the same methods and which, at the same time, would present sufficient characteristics to be significant.

The next table, please. This table indicates the prevalence of pulmonary cancer according to degree of length, to degree and length of exposure and by age. This table shows the prevalence of pulmonary cancer in the group with severe exposure, that's this group, and this is more or less a control group.

This, we have six cases of pulmonary - of primary pulmonary cancer and four cases, in fact, in the group exposed and this, of course, the number being smaller, it gave us a higher incidence, but if we also take that, we have to admit that this difference in incidence is not, has

no statistical value, because it is not more or equal to twice the secondary condition.

Next, please. This table was made at the suggestion of Doctor Pratt, who was interested to know the comparative incidence in group of employee, of employees who have been exposed to a significant degree of exposure, Category 3, 4 and 5, given as less - as one year, and as a control group, we take the group of employees with any kind of exposure, but of the Categories 1, - 0, 1 and 2.

Doctor Pratt had in mind, in suggesting that table, that the factor exposure to severe concentration of asbestos for any length of time might be as significant as the factor asbestosis itself, meaning that if there is a possible causal relationship, the inhalation of sufficient dosage of asbestos fibers might be as important as the factor asbestosis itself and at the same time, I think we have the two extremes.

In the previous slide, we have men with severe exposure, the incidence of cancer, pulmonary cancer in men with severe exposure, and in this - in this table, we have men in the group 2 with very light or very little exposure but with sufficient dosage of - of dust.

Next table, please. This table indicates the prevalence of pulmonary cancer in a series of fifty-three autopsied asbestos workers. This analysis, although not as

important as that of Doctor Merewether of Great Britain, is based on quite a similar basis and I would think that this group is less selective, because that is no section at all. That's all the cases we meant to autopsy after - from fifteen years and over in the industry. Again here, the difference is not as big as it looks, because after using - using the standard there, we came to a difference of about only .5 between the two groups, the group with asbestosis and group with - without asbestosis.

I can not answer why there is less cases of cancer in the asbestotic group, but I think that if we could have autopsy on every employee who happens to die, we might get either a different picture of maybe an indirect explanation.

Next table, please. This table was made for the purpose of knowing if the ratio between the number of cancer deaths outside and the pulmonary cancer deaths was somewhat different from the ratio observed in a few other control groups. First, I think it should be mentioned that ninety percent of the cases for the City of Montreal and a hundred percent of the cases for the City of Thetford Mines, are autopsied, are post mortem cases, so that is why I say it is difficult to compare. I don't know in which proportion for the Province or City of Quebec, but for the Province of Quebec, the statistician and the

epidemiologist at the same time told me that less than ten percent are post-mortem cases, so this might be not comparable to a large extent, but I think if there was any significance, we should expect to have a bigger variation in the percentages.

Next, table, please. Instead of comparing the annual prevalence on incidence of pulmonary cancer in the population of Thetford Mines with the incidence in other control groups, Doctor Phillips from the National Cancer Institute of Canada has suggested to work this number, this table, which is based on the fact that Doctor Domning has found in a random population of 48,833, an annual incidence of seven cases for each hundred thousand of population, given a year expectancy of seventy.

On that basis, Thetford Mines, which is average population of 15,000 for the last ten years, should have ten cases between - twenty-five cases of pulmonary cancer, and the actual prevalence for ten years, this ten years, the actual prevalence is nine cases.

In Montreal, with the ten years expectancy of six, six ninety or six ninety-three cases, it has, as you see, eight hundred fifty-six and I think it is worthwhile to mention that in Montreal, the rate since '45, in '45 the rate per hundred thousand of population was only .5, 5.9, and in 1950, it has reached 12.9.

In conclusion, this paper hasn't been, by any means, the intention to bring the final answer to the problem of the final relation between the inhalation of asbestos fibers and the development of pulmonary cancer, but I think that this incomplete study, I must admit, may have found one of the most representative groups of asbestos workers, believing that the data collected in Thetford Mines do not seem to indicate a causal relationship, and that if it be so, that the data do not correlate the data of Doctor Merewether in Great Britain. Thank you.

(Applause).

BY DOCTOR RHOADS:

I'm curious to observe the remark of Doctor Cartier of the mass survey employed in his group, did not reveal any patients with early pulmonary cancer. I must presume that the mass survey was not sufficiently large, so if this means, after all, for picking up a disease which is treatable and in many instances curable, may not have been sufficient.

I'm also very curious about the fifty-three autopsied individuals of whom ten had pulmonary cancer. I thought they were autopsied because it was thought they may have had pulmonary cancer. It was not a routine autopsy?

BY DOCTOR CARTIER:

No.

BY DOCTOR RHOADS:

are there any questions on this paper? (No response). Of course, the implication is clear that there is no danger of pulmonary cancer from the workers in this particular group, and perhaps you are right. It appears to me that asbestosis is a very healthy ailment as far as cancer is concerned. There seems to be no discussion.

BY DOCTOR PLATTE:

My name is Platte from Kirkman Lake. It's essentially a gold mining district. We now have an asbestos mine in the Town of Banville near Madison. I'd like to ask the doctor what his first signs - I mean it has nothing to do with cancer - but what he looks for for indications of trouble from asbestos?

BY DOCTOR RHOADS:

The question is, what are the first indications of trouble from asbestos exposure. Can you answer that, Doctor?

BY DOCTOR CARTIER:

I don't think there is any specific symptoms except as actually changes in the - if the fibrosis is extensive enough, that is dyspnea and cyanosis, on the extreme degree, but that is, for asbestosis, in my experience, we have had about - I can mention, we have ninety-eight cases of asbestosis in our group and only the group with marked

exposure and many - for many years, and many times when they start to reach fifty-five and over, they are more dyspneac, and I think the main thing, the resistance to it, they seem to die more rapidly once they reach fifty, seventy years without much of any disease. Within a few months, they go down the hill very rapidly.

BY DOCTOR RHOADS:

What do they die of?

BY DOCTOR CARTIER:

They die, some from cardiac and from generalized miscellaneous diseases, and some from bronchiectasis, but I don't think, in my experience, there is any special picture except that in a few, in two or three months, they go down very rapidly.

BY DOCTOR RHOADS:

Doctor Vorwald would like to comment.

BY DOCTOR VORWALD:

I'd like to comment just a bit. We have been encouraging Doctor Cartier to continue or pursue this study in the rather isolated population in Thetford. We are presently conducting a similar epidemiological survey in a neighboring city, also engaged in the mining of asbestos. Of course, I think you realize Doctor Cartier's difficulties in a community such as Thetford, where he must rely upon the material available to him, and we debated how the

incidence of cancer in his exposed population in Thetford might be identified, and we have suggested, as he did, reviewal of the chest Roentgenograms, of all these cases, the Roentgenograms having been taken during the last period of fifteen years. Admittedly though, there will be a number of cases of pulmonary cancer missed by that method. We have considered sputum examination of a large population such as that, but at the moment, the best we can do was survey and study the chest Roentgenograms of this large series of individuals exposed to a special industry.

BY DOCTOR RHOADS:

And you did not pick up any cancer, I take it, in so doing?

BY DOCTOR CARTIER:

Oh, yes, from the X-ray.

BY DOCTOR RHOADS:

From your survey?

BY DOCTOR CARTIER:

Yes - no, not from mass survey; all the cases have been detected in the clinic.

BY DOCTOR RHOADS:

I'm a little puzzled about this. This can be a very interesting topic. You say mass survey didn't do it but the review of Roentgenograms taken when individuals applied for medical care?

BY DOCTOR CARTIER:

No, that's a routine examination at the clinic.

BY DOCTOR RHOADS:

How does that differ from the mass survey?

BY DOCTOR CARTIER:

I think it differs in that the people over fifty years of age do not take advantage of the mass survey, but at the clinic, all the cases have been detected at the clinic and after that they have been followed up, and we have the cases of post mortem.

BY DOCTOR RHOADS:

This indicates what we have referred to before in a number of discussions at this point, that somehow the mass survey doesn't come through to the actual detection and use of the detection facility in bringing the patient into medical hands always.

Any further discussion? (No response). If not, we'll proceed to the next paper. Doctor Kenneth Lynch, as you know, is Professor of Pathology and Dean at the Medical College of South Carolina. Doctor Lynch.

BY DOCTOR LYNCH:

(Doctor Lynch read a prepared paper which is on file at the Saranac Laboratory.)

BY DOCTOR RHOADS:

Thank you, Doctor Lynch, for a very orderly and

Chapter Sixteen

Pulmonary Cancer in Asbestos Workers

Paul Cartier, M.D.

I happen to be in charge of a clinic which.....
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.....any positive evidence.

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SEVEN SARAC SYMPOSIUM - "PULMONARY CANCER IN ASBESTOS WORKERS" PAUL CARTIER

VORWALD COLL.
BOX 91

19pp

MR. CHAIRMAN AND MEMBERS OF THE SYMPOSIUM.

I was asked to present a paper on the topic Pulmonary Cancer in the Asbestos Industry, not on account of any special qualifications but because I happen to be in charge of a Clinic which has as its primary function the supervision of the pulmonary condition of some 4,000 asbestos miners; such a group of employees working in a small radius of 5 miles offers an exceptional opportunity to study the problem of cancer of the lung in relation to the inhalation of asbestos fibers.

PRELIMINARY REMARKS.

This question of pulmonary cancer among the asbestos workers is as confusing and obscure as the etiology and the pathogenicity of cancer in general. From 1920 - 1950 the medical literature reports a series of 58 cases of pulmonary cancer and asbestosis. Obviously this list must be incomplete but, nevertheless, one could ask is it sound to think that the mere fact of detecting a total of 58 cases of pulmonary cancer during a period of 30 years is sufficient to establish a causal relationship when one realizes that in 1950 an estimated number of 45,000 men were employed in the asbestos industry, and that during that period a minimum of 100,000 employees have inhaled a significant amount of asbestos dust?

It is recognized that the English statistics published in the Annual Report of the Chief Inspector of Factories for the

year 1947 seem to indicate a causal relationship between the factor asbestos and pulmonary cancer but, on the other hand, in South Africa with about the same number of asbestos workers as in Great Britain, and when there is also a large number of autopsies and good medical control of all employees in dusty trades, the statistics do not indicate any abnormal incidence of pulmonary cancer.

If the frequency of pulmonary cancer in South Africa and in the Province of Quebec is really not higher amongst the asbestos workers than in any comparable groups as it seems to be, is it possible to suspect that the asbestos mining industry might be different from the Asbestos Textile Industry as far as carcinogenic activity of the asbestos is concerned? The fact that the frequency among the 30,000 asbestos workers of the United States who are also working in the Asbestos Textile Industry is not known to be higher, adds still more to the confusion.

Why is the standardized death rate from cancer of the lungs in Great Britain 3 times higher than that in the United States - 231 in comparison to 723 (rates per million)?

It would indeed be very informative to have some answers to this list of unanswered questions before being able to form an opinion; and it might be that a few answers could influence or modify any conclusion concerning a causal relationship.

PURPOSE OF THIS STUDY.

The purpose of this investigation is to try to learn as precisely as possible the incidence of pulmonary cancer in the group of asbestos mining workers; to know the incidence in groups of employees with severe or insignificant exposure and to know the incidence in groups with or without asbestotic fibrosis.

The Saranac Laboratory and many other investigators were interested in a complete study which would include an epidemiological and an experimental investigation and which would permit to ascertain whether or not a relationship exists between the inhalation of asbestos dust and primary pulmonary cancer. This paper is a preliminary and incomplete report of the epidemiological investigation and the Saranac Laboratory, once the investigation has been achieved, will publish a final report.

METHODS AND MATERIALS.

This study was rendered possible mainly because there exists in Thetford Mines a co-operative industrial clinic where all the employees working in the different asbestos companies are required to have a pre-employment examination, plus an annual periodic examination completed each time by a standard stereogram. At this clinic it was easy to analyse all the medical records, the death certificates, the protocols and the pathological findings of the necropsies and to review the series of chest roentgenograms of each employee. Thetford Mines being a small city of 18,000 it was also easy to contact and to discuss

~~Epidemiology~~ -4- Union Skin

with the treating physician the nature of any respiratory episode, to have follow-up reports on any suspicious cases and to collect additional details on the cause of the deaths.

In summary, this study is based on the results and findings obtained from the analysis of the medical files of 3,957 employees having worked in the asbestos industry from 2 - 60 years, on the findings from 53 necropsies, on the details collected from the death certificates, on the follow-up reports of cases sent to specialized centers for more complete investigation and diagnosis.

It is possible, and more than likely, that cases of pulmonary cancer have been missed; nevertheless, we think that we have employed a standard procedure in this investigation and that the results therefore are of sufficient interest to make this publication worthwhile.

RESULTS.

Before submitting the results of this investigation, it would seem proper to give a few routine tables in order to reach a better understanding of this communication.

TABLE 1.

POPULATION SAMPLED.

Age group distribution of 3,957 asbestos workers.

| AGE GROUPS | <u>15 - 19</u> | <u>20 - 29</u> | <u>30 - 39</u> | <u>40 - 49</u> | <u>50 - 59</u> | <u>60 - 69</u> | <u>70 & Over</u> | <u>TOTAL</u> |
|---------------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------------|--------------|
| Number of Employees | 76 | 1113 | 1091 | 766 | 505 | 307 | 99 | 3957 |

This table gives the age group distribution of the sampled population and needs no comment except to mention that the average age of the asbestos workers is somewhat higher than that of the average industrial worker; i.e., 35 in comparison to 33 for most other industrial groups. This remark is logical when we know that the labor turnover in the Quebec Asbestos Mining Industry is about the lowest of all the industries in the whole of Canada. This first table also shows that some 2,000 asbestos workers are 35 years of age and over - a representative group for the study of a cancer problem.

TABLE 2.

Years employed in the asbestos industry
by 3,957 asbestos workers.

| Years in the Industry | <u>2 - 4.9</u> | <u>5 - 9.9</u> | <u>10 - 14.9</u> | <u>15 - 19.9</u> | <u>20- 24.9</u> | <u>25- 34.9</u> | <u>35 & Over</u> | <u>TOTAL</u> |
|--------------------------|----------------|----------------|------------------|------------------|-----------------|-----------------|----------------------|--------------|
| Number of Employees | 640 | 1060 | 645 | 476 | 281 | 546 | 309 | 3957 |
| Man - Years | 1920 | 7420 | 7740 | 8092 | 6182 | 14742 | 12360 | 58456 |

REMARKS: 2,257 men have spent 10 years and over in the asbestos industry.

The second table indicates the number of years spent in the asbestos mining industry by the 3,957 employees studied. Of course this table is not as significant as one indicating the specific exposure for each employee by categories; but after many attempts it has seemed impossible to find a common denominator which would permit to add together the years spent in different degrees of exposure by the same employee.

The man-years exposure has in the mind of many investigators no great meaning in general, but in this case the fact that 56,456 man-years asbestos exposure having given rise to only 10 confirmed cases of pulmonary cancer might indicate that we are not facing an acute problem.

The following table 3 shows classification of various exposures in the asbestos mining industry. I realize that such a classification could be discussed at length and possibly

modified because in the asbestos industry, as in most of the other dusty industries, it is impossible to translate with a strict mathematical precision the different concentrations of dust particular to each job. This classification is given notwithstanding its limitations because it is based on dust counts performed by different competent agencies and, moreover, because we feel that a satisfactory classification is very useful in understanding more clearly scientific and medico-legal problems.

TABLE 3.

Classification of various exposure in the asbestos mining industry.

| Estimated degree of hazard. | Industrial Operations | ATMOSPHERIC CONDITIONS | |
|-----------------------------|--|---|-------------------------|
| | | Total dust count M.P.C.F. of air sampled. | Percent fiber count. |
| 0 | Open Pit, Shops | Less than 1 | Less than 2 |
| 1 | Open Pit: Box Loader, Cobber Dump | 1 - 3 | Less than 2 |
| | Primary Crusher | 1 - 3 | Less than 2 |
| | Dryer | 2 - 3 | 5 |
| | Concentrating | 2 - 6 | 6 |
| | Underground: all jobs under wet condition. | 2 - 5 | 5 |
| | Short exposure of skilled employees | 3 - 6 | 1 - 2 |
| 2 | Open Pit: dry drilling | 2 - 5 | 1 - 2 |
| | Secondary Crushing | 10 - 12 | 4 - 6 |
| | Storage Bin | 10 - 14 | 4 - 6 |
| 3 | Underground: dry drilling | 10 - 15 | 1 - 3 |
| | Grizzlies | 5 - 15 | 2 |
| | Shed Cobbing | 3 - 5 | 14 - 16 |
| | Shipping Shed: Cleaners, Pilers | 4 - 15 | 10 - 14 |
| 4 | Mill: Millwrights, Oilers, Sweepers. | 10 - 25 | 25 - 9 |
| | Testers. | 8 | 20 |
| 5 | Bagging Department | 20 - 30 | 28 |

This table indicates that the total dust count and the percent fiber count are both constituents of the degree of hazard, meaning that a rather low total dust count may become a risk when the percentages of fibers is high and inversely, that a high total dust count may also become harmless in producing pulmonary fibrosis if the fiber percent count is low.

The percentage of fibers in relation to the total count is calculated for fibers 5 microns and over, and on the principle that only particules having a length at least twice its width are fibers.

As you may see in the table, the industrial operations are grouped in 6 different categories and these categories are again sub-divided by a double line in two main groups. The upper group includes the jobs which in our experience do not produce fibrosis of some clinical importance and the lower group the jobs which can produce asbestosis.

There is an obvious explanation why the men working in the open pit do not develop asbestosis and also why the men working as baggers might develop asbestosis, but it is more difficult to explain why some industrial operations with about the same dust concentration as secondary crushing, storage bin on one part, the grizzlies and shed cobbing on the other part have been classified in different categories. We admit that in adopting such a classification we have been influenced by our clinical findings to some extent, but also by the fact that in the operations of category 3, the fiber concentration is more constant and work much heavier than in the operations of category 2. There is a marked difference

in the degree of hazard of categories 1,2 and 4 and 5, but the difference is not so marked for the categories 2 and 3. This table is important in order to fully understand those to follow.

It is true that the main purpose of this presentation is to report the incidence of pulmonary cancer in a group of 3,957 asbestos workers, but the nude statement that 10 cases of pulmonary cancer have been detected would not be significant unless we can compare this prevalence with that in comparable groups.

This task of finding a good comparable group or a control group in this study as in any similar studies is very difficult. We wish it had been possible to find a control group in other industries or in other industrial centers in the Province of Quebec or elsewhere in Canada. We soon realized that no other industries were interested in that problem of cancer of the lungs nor did they have the necessary data for comparison. No other industrial city has ever had two-thirds of its male adult population of the same age group so closely medically supervised with a clinical and routine roentgenographic examination as has been done in Thetford Mines for the last 7 years. The mass surveys in different cities did not help the situation; the findings were too dificient doubtless because a too small proportion in the 40-year and over age group is taking advantage of the mass survey. As a matter of fact, a mass survey in Thetford Mines in 1950 by the Provincial Department of Health did not detect a single case of pulmonary cancer as in most of the other centers, and the analysis of the results is not very helpful.

It seems probable that the general increase in the frequency of lung cancer is not purely apparent, but when we observe in many cities a sudden and important increase in the lung cancer mortality rates such as the increase in the City of Montreal from 7.7 in 1946 to 12.9 in 1949, we must admit that the medical diagnosis facilities are responsible to an appreciable extent for such an increase. Therefore the exclusive analysis of death certificates in various centers which do not have the same medical facilities, has to be used with caution not should it be used unless the facilities are comparable. In this study the comparative statistical analysis of the death certificates was not used because in the opinion of the official statistician of the City of Montreal and of the Statistician of the National Cancer Institute of Canada, the death certificates of the asbestos group consisted of a highly selective group and a similar selective group was not available elsewhere.

For all the reasons mentioned above and the fact of being unable to make a better selection, we decided to compare the incidence of pulmonary cancer in different groups of asbestos workers who have been submitted to the same medical investigation, and who at the same time were presenting enough characteristics to permit certain conclusions or impressions.

TABLE 4.

Incidence of pulmonary cancer
according to degree and length of exposure by
age groups.

| | <u>GROUP 1.</u> | | <u>GROUP 2.</u> | |
|--------------------------------|------------------------|-----------|----------------------|-----------|
| | Unsignificant exposure | | Significant exposure | |
| | AGE GROUPS | | AGE GROUPS | |
| | 16 - 39 | 40 - 86 | 16 - 39 | 40 - 86 |
| Number of Employees | 2229 | 1367 | 56 | 305 |
| Prevalence of Pulmonary Cancer | 0 | 6 (0.44%) | 0 | 4 (1.31%) |

REMARKS: Unsignificant exposure includes men with exposure 0-1-2 and less than 15 years 3-4-5.

Significant exposure includes only men with more than 15 years 3-4-5. (See Table 3)

The usual calculations of the standard error show that the observed difference in percentages of incidence in the 2 groups (.87) is inferior to twice the Standard Error (1.346).

This table shows that there is no statistical evidence that the incidence of pulmonary cancer in the group 2 including the employees who have sustained severe exposure and all those who have also developed asbestosis, is higher than in group 1 of employees those without significant exposure and without any clinical asbestosis.

It is recognized that this table might lose a large part of its meaning if it is shown that any intensity of asbestos inhalation or any amount of asbestotic fibrosis could be considered as cancerigenic because then a large number of employees in group 1 will be transferred to group 2 and it is probable that an unknown number of pulmonary cancers in the group 1 would also have to be transported to group 2, changing rapidly the observed difference in incidence as well the conclusion.

As a logical complement to this table, we wish we could give a long series of other tables showing the incidence of pulmonary cancer in groups with recent or remote exposure; in groups with gradual exposure of 1-2 to 15 years or a similar exposure of long duration, 15 years and over; also in groups with extensive exposure and asbestosis or without asbestosis.

The data obtained from such tables might tell us in the hypothesis of a causal relationship whether the inhalation of asbestos dust is more important than the presence of asbestosis itself also what approximative degree of asbestos inhalation can be considered as predisposing to cancer.

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TABLE 5.

Comparative Incidence of Pulmonary
Cancer in 2 groups of 53 Autopsied asbestos workers.

| | GROUP 1. | GROUP 2. | TOTAL |
|----------------------------------|------------------------------|---------------------------------|-------|
| | Employees with Asbestosis | Employees without Asbestosis | |
| Number Autopsied | 34 | 19 | 53 |
| Number of Pulmonary Cancer | 4 | 6 | 10 |
| Percent in Each Group | 11.7% | 31.5% | |

This table shows the prevalence of pulmonary cancer in a series of 53 autopsied asbestos workers having worked in the asbestos mining industry from 17 to 40 years in different degrees of exposure.

This series of autopsies is not as important as the series published in the Annual Report of Great Britain for the year 1947, but can be considered as less selective and more representative in not being composed from cases of asbestosis exclusively but composed of all the employees who happen to be autopsied; although it should be mentioned that there was an

involuntary selection in the sense that most of these employees have been autopsied because they were presenting some respiratory symptoms.

This coincidental selection might explain to some extent the high percentage of cases of asbestosis, but in many instances the amount of asbestotic fibrosis detected was described as minimal and of no clinical importance because not sufficient to produce symptoms.

Because the observed difference in percentages of incidence is inferior to twice the Standard Error, this table does not reveal a statistical evidence of a causal relationship between asbestosis and pulmonary cancer.

Unless we have the opportunity to perform an autopsy on all employees who die, it remains difficult to explain why in our series there are less cases of pulmonary cancer in the asbestotic group and I think we have here a good illustration as to why many statisticians counsel not to draw any conclusions from statistics based on a selective group even if such a group be in the 'autopsy' category because the role played by the factor co-incidence might be too great.

TABLE 6.

Deaths from Pulmonary Cancer as a proportion
of deaths from Cancer of all sites.
1940 - 1950

| | Number of Cancer All forms | Number of Pulmonary Cancer | Percentages |
|---------------------------|-------------------------------|-------------------------------|-------------|
| Province of Quebec | 43643 | 1393 | 3.2 |
| City of Montreal | 15247 | 918 | 6.0 |
| City of Quebec | 2543 | 94 | 3.7 |
| City of Thetford Mines | 172 | 9 | 5.2 |

Table 6 was prepared for the purpose of determining if the ratio between the number of cancer deaths all sites and the pulmonary cancer deaths was somewhat different in Thetford Mines from the ratio observed in few other control groups. First it should be mentioned that about 90% of the cases of pulmonary cancer in Montreal and 100% of the cases of the Thetford Group are autopsy cases and both cities have thoracic clinics, so these two groups are reasonably comparable. In Quebec City for the specific period 1945 - 1950 inclusive, 5% of all cancer deaths were also pulmonary cancer. It was thought that had a special factor existed in Thetford Mines the ratio between the number of total cancer and the number of pulmonary cancer would have been different. The difference observed here is not sufficient to draw any conclusion either positively or negatively.

TABLE 7.

Comparative 10 Years Expectancy and 10 Years
Prevalence of Pulmonary Cancer in 2 cities.

| PLACE | YEARS | 10 YEARS EXPECTANCY | 10 YEARS PREVALENCE |
|----------------|-------------|------------------------|------------------------|
| Thetford Mines | 1941 - 1950 | 10.5 | 9 |
| Montreal | 1941 - 1950 | 693 | 856 |

Based on U.S. Public Health Service Report 2537. A random population of 48,833 giving a year pulmonary cancer expectancy of 7 cases per 100,000 population.

Table 7 was prepared at the suggestion of Dr. Philipps, Statistician for the National Cancer Institute of Canada who thinks that the comparative study of the 10 years expectancy and 10 years actual prevalence of pulmonary cancer in Thetford Mines and in Montreal will be more informative and more conclusive than analysis of the mortality rates. According to the survey of Dr. Dorn, taking random population of 48,833, an annual incidence of pulmonary cancer of 7 cases per 100,000 can be expected, giving a year expectancy of 7. It is true that Dr. Dorn's survey was made in 1943-45 and that the year expectancy might have increased recently. As a matter of fact, Watson of Saskatoon, Canada, found in 1948 a year expectancy of 8.5; therefore, a year expectancy of 7 is a rather conservative standard.

On that basis Thetford Mines with an average population of 15,000 for 1940 - 1950 period is supposed to have 10.5 cases

of pulmonary cancer, and the actual prevalence is 9 cases. Again it seems logical to think that the 10 year prevalence for pulmonary cancer in Thetford Mines where two-thirds of its adult population is working in the asbestos industry would be higher than the normal 10 year expectancy, should the factor asbestosis be considered carcinogenic.

SUMMARY.

The etiology and the pathogenicity of the pulmonary cancer in relation to asbestos factor is as obscure and confusing as those of cancer in general, and in presence of contradictory data, the many different aspects of the problem should be considered before arriving at a conclusion.

The purpose of this study is to learn the incidence of pulmonary cancer amongst the 3,957 asbestos mining workers and to compare this incidence with other comparable groups. This incomplete epidemiological report is a contribution to a more complete investigation under progress at the Saranac Laboratory.

According to the standard procedure the medical files, the results of medical supervision, the annual stereograms of all the asbestos workers having worked from 2 to 60 years and the findings of 53 autopsies have been used as material.

The results have been tabulated and compared to control groups and we think that the data collected in the Asbestos Mining Industry are not exactly superposable to the results found in the

English Asbestos Textile Industry of Great Britain and if they do not permit to deny a causal relationship between the inhalation of asbestos dust and the occurrence of pulmonary cancer, they do not seem either to bring any positive evidence.

Exhibit

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Discussion

DR. RHOADS: I was interested in Dr. Cartier's remark that the mass survey employed in his group did not reveal any patients with early pulmonary cancer. I presume that the mass survey was not sufficiently large and therefore this method of revealing a disease which is treatable and often curable may not have been adequate.

— In regard to the 53 autopsied individuals, of whom 10 had pulmonary cancer, I presume they were autopsied because it was thought that they may have had pulmonary cancer. Those were not routine autopsies?

DR. CARTIER: No.

DR. RHOADS: The implication is clear that there is no danger from pulmonary cancer to workers in this particular group. Perhaps that inference is true. It appears to me that asbestosis is a very healthy ailment as far as cancer is concerned.

UNIDENTIFIED SPEAKER: Dr. Cartier, what are the first signs of trouble from exposure to asbestos?

DR. CARTIER: I do not believe there are any specific symptoms. If the fibrosis is extensive enough there is dyspnea and cyanosis, sometimes to an extreme degree. In our group we have had 98 cases of asbestosis. These individuals have had a marked exposure to asbestos for years and many of them, on reaching age 50 to 55, are more dyspneic and seem to die more rapidly without much disease. They go downhill rapidly, within a few months.

DR. RHOADS: Of what do they die?

DR. CARTIER: Some from cardiac disease and from miscellaneous generalized diseases, some from bronchiectasis. In my experience there is no special picture except that they fail rapidly - in two or three months.

DR. VORWALD: We have been encouraging Dr. Cartier to continue this study of the rather isolated population of Thetford, and we are conducting a similar epidemiological survey of a neighboring community which is also engaged in the mining and processing of asbestos. We suggested to Dr. Cartier that he might determine the incidence of cancer in his exposed group in Thetford by reviewing the chest roentgenograms of his cases - the roentgenograms taken during the past 15 years. Admittedly, cases of pulmonary cancer will be missed by that method. We have considered sputum examinations but at present the best we can do is to study the chest roentgenograms of this large series of individuals exposed in a special industry.

DR. RHOADS: Did you discover any cancer cases, in so doing?

DR. CARTIER: Yes, with the X-ray. No cases were revealed by the mass survey; all the cases were detected in the clinic.

BY DOCTOR RHOADES:

Thank you, Doctor Lynch, for a very orderly and

Chapter Seventeen

Carcinoma of the Lungs in Asbestos Weavers

Kenneth M. Lynch, M.D.

in
Book

Perhaps the main justification.....
.....factor were uncovered.

CARCINOMA OF THE LUNGS IN ASBESTOS WEAVERS

BY

KENNETH M. LYNCH, M. D.
Professor of Pathology
Medical College of South Carolina
Charleston

(Summary of presentation at the Seventh Saranac Symposium,
Saranac, New York, September 24, 1952)

Perhaps the main justification for my acceptance of the invitation to discuss the subject of this paper in this company rests in my interest in the questions concerned more than in any special contribution that I might make out of a rather sketchy and spasmodic indulgence in efforts to solve them. I am sure that others who are present here are better qualified, and my purpose is to gain from them rather than to expect them to benefit from any offering of mine.

When in 1934 I had the opportunity to examine by necropsy the case of carcinoma of the lung that was to become apparently the first of such an occurrence in a case of asbestosis to be formally set into published records¹, there immediately arose the question of a possible causal relationship in the prior state of chronic lung disease. We had for several years been deeply interested in asbestosis, and this new occurrence produced an additional question for thought and investigation.

At least up to that time chronic inflammatory disease, or chronic irritation, particularly with fibrous production and disarrangement of structure, was the main anchor in speculations / ^{concerning the} etiology of

cancer, particularly of epithelial types. In advanced asbestosis an opportunity for the occurrence of carcinoma of the lung on that basis seemed to be good. Actually nothing has yet occurred to cast out that possibility.

In the intervening time, during which we were to encounter two other like cases, there appeared scattered reports of the same occurrence in other locations; until, by compilation in 1947 Merewether² states that there were then recorded at least 31 instances of cancer of the lung in 235 cases of asbestosis, an autopsy pulmonary cancer incidence of 13.2% in persons suffering from asbestosis.

For a fully acceptable comparison of this incidence of course one would be limited to the use of an autopsy series from a closely similar group in age and sex and at least in like environment for a similar period of time except for exposure to asbestos inhalation. Even these rather loosely set criteria could hardly be met where numbers would be large enough to provide ^{only} a minimal chance of error in statistician-type calculation.

The most that can be done at the present is to take what is available as evidence for or against an etiological basis for cancer of the lung in asbestosis, at the same time recognizing that the large chance of statistical error does not allow proof to be claimed but merely suspicion to be supported or not supported.

At present such evidence as is available appears to support the suspicion and to justify further investigative effort of whatever nature may be scientifically ^{or soundly} devised. At least the available evidence does not allay the suspicion.

In South Carolina the autopsy service that has furnished the human material for our studies in asbestosis is in a general hospital that through the years might be expected to produce a cross section of the run of serious diseases of the community of location.

For such value as it may have we have analyzed the findings in the 3943 autopsies occurring in that service from 1926 to 1950 in the age period of twenty years or over, the purpose being to cover a span of twenty-five years that corresponds roughly to the period of observation of asbestosis and an age group that would also cover the occurrences of that condition.

In that total autopsy series were found 46 instances of a diagnosis of asbestosis by microscopic evidence, including all cases regardless of degree, and among these 46 were three with advanced carcinoma of the lung, an incidence of 6.5%. In all others carcinoma of the lung occurred 58 times, an incidence of 1.5%.

Obviously such a comparison is of limited value, as are all others from which the question has been posed, although taken as a whole they are at least in agreement that carcinoma of the lung has been consistently more common than was to be expected in reports on asbestosis. Our incidence of that order is less than in some other reports, while our general incidence of carcinoma of the lung is comparable to some (Homburger) but less than others (Rigdon & Kirchoff). Our incidence in asbestosis would approach that given in other reports if the calculation were made within the group of medium and advanced grades of asbestosis, in which area the three instances of pulmonary carcinoma occurred. In that group we had 28 cases, and the incidence within the group would be 10 4/5%.

Incidentally, for such support as it may give to the common report of growing prevalence of carcinoma of the lung, we find that in the first third of the time period covered in our series the incidence of carcinoma of the lung was 0.9%, in the second third 1.1% and in the latest third 2.2%, apparently a steady increase during the twenty-five year period, even though still not as high as reported from some other areas.

Without at this time attempting to make a thorough analysis of the published papers and records, a few recent pertinent reports are noted.

In one of the latest, Stoll, Bass and Angrist³ report an additional case of cancer of the lung in association with asbestosis and cast up the literature as of 1951. They refer to Merewether's 1947 report, as previously noted herein, and also refer to a report by Wyers in 1949 recording a series of 115 cases of asbestosis in which there were 17 instances of associated pulmonary carcinoma, an autopsy series incidence of 14.8%, as compared to Merewether's 13.2%.

Homburger⁴ reported three additional cases of association of pulmonary carcinoma and asbestosis in his autopsy service. In his series of autopsies from 1918-1938 there were 45 cases of pulmonary carcinoma, an incidence of 1.08%. Asbestosis was diagnosed 8 times and in 4 of the 8 cases pulmonary carcinoma was also found.

For such comparison as may be allowable, Rigdon and Kirchoff⁵ reported in 1951 a pulmonary cancer incidence of 7.5% (67 in 892) in an autopsy series in individuals 30 years of age and older in Texas. They suggest "that frequency of cancer of the lung/^{would best} be established from a series of autopsies," on account of the unreliability of death certificates.

Counterbalancing at least some of the weight of evidence of reports of cancer of the lung associated with asbestosis is the widespread increase of pulmonary cancer in the general population that has been recognized during the same period of time. A few references will serve to focus that tremendous problem.

Graham⁶, in 1951, says, "Bronchogenic carcinoma differs from other cancers in two important respects: (1) it has shown an enormous increase in the last 35 years; (2) there has been a progressively larger incidence in the male sex."

He states that Adler in 1912 could collect only 374 cases of cancer of the lung from the world's literature, while individual experiences in this country greatly exceed that total of reported cases from the whole world, and that cancer of the lung has advanced to a position of first place in men from one of 8th place in frequency - and this all within the last 35 or 40 years.

Doll and Hill⁷ refer to a British government report which shows that for the 25 year period from 1922 to 1947 the incidence of lung cancer in autopsies in England and Wales increased fifteen times over what it had been previously.

An editorial note in The Lancet in January, 1952, strikes a timely note in referring to Hill's report that "in at least 17 out of 29 boroughs (metropolitan), deaths from neoplasm of the lung and bronchus were more numerous than deaths from tuberculosis" and in remarking, "This is something quite new."

Smith⁸, in 1952, states that lung cancer has shown an increase over and above what could be expected as a result of increases in numbers of elderly persons. He states also that high lung mortality has been recorded for both men and women patients with asbestosis in England.

Perhaps a personal note is permissible to illustrate the apparently universal experience in autopsy services during the last forty years that carcinoma of the lung has changed from a rarity to a commonplace. As a resident in the Philadelphia General Hospital

I had done an autopsy on what I took to be a case of tuberculosis, and had discarded all organs and tissues except the small pieces from which I prepared microscopic sections. When I showed the resulting slides of carcinoma of the lung to my chief and had to acknowledge that I had thrown the lungs away, he arose sternly from his microscope and literally booted me down the stairs.

Perhaps I may also note that the scarcity of residents in pathology nowadays has ~~also~~ brought about a difference in ^{their} treatment, although possibly not so much difference in their deserts.

EXPERIMENTAL STUDIES

Because of the question raised by autopsy observation of associated asbestosis and cancer of the lung, and among the many experimental studies that ^{apparently} have been done and ~~for~~ are now going on by subjection of experimental animals to respiratory exposure to a variety of suspected substances, I have undertaken to see whether evidence of carcinogenic action on the part of asbestos or of the condition of asbestosis might be gotten in this way. Now in the third year, we have such a project under way, supported in part by a National Cancer Institute grant.

In devising such an experiment, one is, of course, faced with many questions, among which are the choice of experimental animals, the methods of exposure, the mechanical questions involved in methods, the practical matters of time and help, etc., etc.

Because, so far as I am aware, no animal except man has shown bronchogenic carcinoma of the lung naturally (or experimentally for that matter)

we chose to work with a strain of mice known to develop adenomatous tumors of the lung in regular pre-calculable occurrence. In this animal we have one of short life span, of known lung tumor productiveness that might be susceptible to influence in that natural occurrence, and at the same time as good as any other prospect for bronchogenic carcinoma so far as is known. The strain chosen is the AC₁ hybrid (A male and C female) first generation from breeding stock obtained from the Roscoe B. Jackson Memorial Laboratory. Approximately equal numbers of mice are being exposed and kept as controls. Exposure chambers are of our own devising. Regular dust counts are made, with concentrations of dust as high as may be ^{obtained} without risk of killing the animals. As near as feasible, the same asbestos as our human cases experienced is being used as dust.

As the animals are sacrificed they are autopsied and the number, size, and distribution of lung tumors recorded. An attempt to grade these will also be made. It would be premature to give any analysis of this data as yet. However, no epidermoid carcinoma has yet been observed.

Because we wished to also use another and larger animal, a number of dogs are ~~also~~ being subjected to similar dusting in specially constructed chambers. This is, of course, a much longer term experiment, and no report can be made upon it at this time. One of the reasons the dog was selected is that observation of the occurrence of asbestosis bodies in the lung of a dog exposed in an asbestos factory is recorded.

So far as I am aware, there is only one published report of an inhalation experiment exposing mice to asbestos dust as a test

of cancerigenic action. This report, by Nordmann and Sorge,⁹
by the authors
was claimed/as positive evidence of such a relationship and has
been cited to that effect by others. Smith,⁸ however, analyzes
it to the conclusion that it not only produces no proof of a
causal relationship of asbestos in cancer of the lung but actually
to the contrary. In that report ill-defined epithelial changes
were thought to have resulted in the lungs of 9 animals out of an
original 100 mice, squamous metaplasia was found in 6, adenocar-
cinoma in one and squamous cell carcinoma in one. Smith questions
whether the interpretation was correct as to the demonstration of
actual squamous cell carcinoma and calls attention to the common
occurrence of squamous cell metaplasia in old rats with chronic
pulmonary infections and in animals deficient in vitamin A.

The only other experiment of which I am aware where animals
under exposure to inhalation of asbestos dust developed tumors
of the lung in a way suggestive of a relationship occurred in an
uncompleted experiment of the late L. U. Gardner that came to my
attention through a courtesy from the Saranac Laboratory.

In one of Gardner's experiments in asbestosis he found a
high incidence of the type of lung cancer to which mice are disposed.
Since the strain he was using was unknown as to tumor susceptibility
and since the experiment was uncontrolled from that standpoint, he
stopped it, apparently intending to set up a proper experiment to
follow up the suggestion later.

At least it does not appear that any reported experiment can
be accepted as conclusive, and I am inclined to the same view as
expressed by Smith, that up to the present no experimental proof
that the inhalation of asbestos or any other material will cause

bronchogenic carcinoma has been presented.

As the matter now stands, it appears that while additional evidence of the same sort has been collected in the meantime, the question that was raised with our first report in 1935 as to whether asbestosis can act as a producer of cancer of the lung in human beings remains as open as it was when raised.

So far as we are concerned, the conditions in the industry are so much improved that even the problem of asbestosis has become greatly lessened. The grade of exposure to the dust has been so reduced in our territory of experience that we no longer expect to see the extremes of the disease that earlier uncontrolled conditions caused.

Even so, and although our cases were those of ^{medium to} advanced asbestosis, if the exposure may be cancerigenic it could not now be certainly said that lesser grades of the primary disease would be immune to the tendency.

In all respects, therefore, the problem remains with us and should be subjected to continued investigation on its own merits, aside from any advantage that would accrue to the investigation of malignant neoplastic disease as a whole if such an etiologic factor were uncovered.

REFERENCES

1. Lynch, K. M. and Smith, W. A.: Pulmonary Asbestosis III: Carcinoma of Lung in Asbesto-Silicosis. *Am. J. Cancer*, Vol. XXIV, No. 1, May, 1935.
2. Merewether, E. R. A.: Annual Report of the Chief Inspector of Factories, London, His Majesty's Stationery Office, 1947.
3. Stoll, Roger; Bass, Richard; and Angrist, Alfred A.: Asbestosis Associated with Bronchogenic Carcinoma. *Arch. Int. Med.*, 88, 831 (Dec.) 1951.
4. Homburger, F.: The Coincidence of Primary Carcinoma of the Lungs and Pulmonary Asbestosis. *Am. J. Path.*, 19:1943, 797-805.
5. Rigdon, R. H. and Kirchoff, Helen: Frequency of Cancer of the Lung in All Malignancies Studied at Autopsy. *Sou. Med. J.* 44:506, June 1951.
6. Graham, E. A.: Pulmonary Cancer of the Lung with Special Consideration of its Etiology. *Bull. N. Y. Acad. Med.*, 27:261, May 1951.
7. Doll, Richard and Hill, A. Bradford: Smoking and Carcinoma of the Lung. *Brit. Med. J.*, #4682, 739-748, Sept. 30, 1950.
8. Smith, W. E.: Lung Cancer With Special Reference to Experimental Aspects. *Arch. Ind. Hyg. & Occup. Med.*, Vol. 5, No. 3, 209, March 1952.
9. Nordmann, M., and Sorge, A.: Lungenkrebs durch Asbeststaub im Tierversuch, *Ztschr. Krebsforsch.* 51:168-182, 1941.

interesting presentation. I hoped to be able to make a pertinent comment, but Doctor Lynch disposed of that in the last part of the talk. I gather there is no correlation between the extent of asbestosis in your series and the occurrence of neoplastic change?

BY DOCTOR LYNCH:

In our series, Doctor Rhoads, the three cases, now, remember there are only three - occurred in advanced cases of asbestosis, not in minor conditions where you merely make a diagnosis because the asbestosis might be present.

BY DOCTOR RHOADS:

I would ask those who are going out if they wouldn't mind waiting until we take the photographs in just a few minutes. Doctor Vorwald, we're anxious to keep all possible here for the photograph.

Is there other discussion on Doctor Lynch's paper? I hear no discussion. Doctor Vorwald and I both feel, because of the time and the need for taking the photograph, we had best postpone Doctor Merewether's paper until after lunch. Doctor Vorwald, do you care to speak about the photograph?

BY DOCTOR VORWALD:

Yes, we would like to take a photograph of the group as we are seated here. Is the photographer here?

(No response).

Now, if those of you who are in the back, could you come forward so that we can really get a picture. There are a lot of seats up forward and it will only take a few minutes.

I might say that the photographer will print these pictures and you may buy one from him if you so wish; on the bulletin board outside will be a paper where you might indicate whether you want one or a dozen.

(Photograph was taken, after which the meeting was adjourned. Session reconvened at 2:37 P. M.).

PNEUMOCONIOSIS AND PULMONARY CANCER (Continued) 2:30 to
5:30 P.M.

Chromate Workers

Anthony J. Lanza, M. D.

Discussion

Experimental Pulmonary Cancer - General Review

G. Burroughs Mider, M. D.

Discussion

Pulmonary Cancer in Experimental Exposures to Chromate

Anna Baetjer, Sc.D.

Discussion

Pulmonary Cancer in Experimental Exposures to Beryllium

Arthur J. Vorwald, M. D.

Discussion.

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Discussion

Chapter Eighteen

Pulmonary Cancer in Chromate Workers

A. J. Lanza, M.D.

The available evidence is that chromate workers.....

.....

.....heading up our program.

see
page
74-A
Lanza

The available evidence is that chromate workers do have an incidence of cancer of the ^{lung} in excess of what might be expected. Beyond this observation, we do not as yet know a great deal. Interest in chromate as a possible carcinogenic agent, with particular reference to lung cancer, is comparatively recent. The officials of one of the largest chromate producing companies in this country supplied the impetus that started interest in this subject in the United States by setting on foot the statistical survey conducted by Macchic and Gregories and published in the Public Health Reports of August 27, 1948. Efforts were made to reduce exposure in chromate plants and a more intensive medical service was inaugurated, including much greater use of X-ray examination.

An investigation of the problem was carried on in Baltimore by Dr. Baetjer with experimental animals. In 1951 she published ^{in the Archives of Industrial Hygiene and Occupational Medicine,} a comprehensive paper on pulmonary carcinoma in chromate workers and, up to now, there is not much more to say that was not said in her paper. In a word, she covered the subject completely.

In 1953 the Division of Occupational Health of the U.S. Public Health Service, with the assistance and cooperation of the industry, conducted a survey of the whole chromate industry through the courtesy of Dr. Edward S. Miller, in charge of the Division of Occupational Health, who has given much of the medical work on the admirable report (Health of Chromate Workers, pub

the report:

For all specific causes of mortality, except cancer, in chrome workers, the chrome workers had frequency rates that were not greatly different from those for other workers. However, cancer with a rate of 7.1 compared with 2.7 stands out as markedly in excess for chromate workers. For cancer of all sites the actual number of deaths of chromate workers was approximately ~~four and a half~~^{4 1/2} times the number that would have been expected had the cancer rate for all males in the United States prevailed. When cancer of the respiratory system was observed separately for chromate workers, nearly 29 times as many deaths as were expected were found. All other types of cancer failed to show an excess among chromate workers.

It is obvious that much research and experimentation remain to be done before we can talk with more assurance of this situation. In the meantime the industrial plants are being cleaned up, and in some cases being rebuilt, with the hope and expectation of stemming this tide of cancer cases.

Before leaving this topic I would like to make a plea again for the more careful and precise use and association of words.

The evidence of the relationship of cancer and chromate is conclusive. This cancer of the lungs in chrome workers does not follow, nor is it dependent upon, smoking. I mention this so that there will not be any

Discussion

DR. RICHARDSON: Which workers of those included in the survey were affected?

DR. LANZA: I cannot tell you positively until I have studied the Public Health Service report. The plants are small, the men shift around, and the details of their exposure to chrome are rather indefinite. *We do not know what particular substance or what particular process the manufacture of chrome may be responsible.*

DR. RICHARDSON: Could one say just when the exposure to chrome begins and when it ends in the process of handling the chrome?

DR. LANZA: The engineering portion of the Public Health Service report gives the details of the process and the dust studies at various operations from the time the ore is received in the plant until it is shipped out.

DR. VICKERS: Workers in the chrome industry are exposed to chromate in one way or another all through the plant.

DR. RHODES: The exposure is not limited to any particular operation or to any particular form of chromium?

DR. VICKERS: No. There are mixed exposures of one kind or another, but definitely to chromium, all the way through the plant.

Richardson?
DR. RICHARDS: Is there any hazard after the material has been shipped out?

DR. LANZA: So far as I know, there has been no complaint.

DR. HUEPER: In regard to the possibility that the material shipped out of the plant will become carcinogenic, there have been reports from Germany that zinc pigment - zinc chromate - workers have developed carcinoma. We are attempting to find out whether the painters who sprayed zinc chromate on airplanes during the last war have an increased cancer incidence.

DR. BAETJER.

DR. Baetjer

One hundred thirty-four cases of carcinoma of the respiratory tract have been described in the literature among men exposed to chromium chemicals in their occupation. Of that number 121 had worked in the chromate chemical manufacturing industry and 11 in the chrome pigment industry. Only 2 of the men had been employed in industries which use chromium compounds, one of which was a chrome plater and one had sprayed chromium paint.

We have never ^{heard of any cases of} ~~seen~~ cancer of the skin in chromate workers [✓] for cancer of the nasal septum even though a large percentage of the workers have perforation of the nasal septum ^{and ulcers of the skin due to the chromium chemicals.}

It is interesting to review the duration of exposure of these people to chromium before they ^{developed} ~~get~~ cancer. The average age of these individuals ^{at the time of death} was 52 years and the range of ages was from ²⁹ ~~31~~ to 72 years. The duration of exposure varied from 4 to 47 years ^{with an average of 18 years} ~~and the interval between the initial exposure and the diagnosis of cancer was about 17 years, with a range of 4 to 47 years.~~ In some cases cancer may develop many years after the termination of exposure but the interval between diagnosis and death is, as in all cases of lung cancer,

usually a very short period of time. ^{Some} Many of our cases, on coming to ^{the Hospital} our dispensary, claimed that their severe symptoms didn't begin until two weeks before ~~these individuals~~ ^{they} came to the clinic, and they ~~could then be~~ ^{were} diagnosed as having lung cancer. So far as we have been able to determine, the clinical symptoms and the pathology are no different for lung cancer in chromate workers than for lung cancer of other origin.

DR. RHOADS: What brought these patients to the clinic? Symptoms referable to the lungs?

DR. BAETJER: Some came with lung symptoms ^{such as} ~~the~~ pain in the chest; ^{the} some patients about whom I have read came with symptoms referable to ^{the} metastases.

See further
Discussion
November 16

Corrected
by
Cruik

BY DOCTOR RHOADS:

Will the meeting please come to order? You will remember that we were forced by the delay starting this morning, to put over Doctor Merewether's paper to the first one on the program this afternoon. It is, therefore, my honor to introduce E. R. A. Merewether, Senior Medical Inspector of Factories, Administrator of Labor and Allied Service, London, England. Doctor Merewether.

BY DOCTOR MEREWETHER:

Doctor Rhoads, Ladies and Gentlemen: I am greatly honored to be invited to attend this erudite meeting which has now, by virtue of the diverse and inspired work of my old friend, the late Doctor LeRoy Gardner, and Doctor Vorwald, become an international event.

Now, only a foolhardy criminal would speak after the most stimulating papers of this morning, but I'll endeavor to do my best, and I shall not detain you long.

All I have been able to do in the short time available is to bring up to date our mortality and pathological data on this matter and lay them before you in the confident hope that if there is an explanation of them, and there are deductions to be drawn from them, I shall obtain them during this conference.

Frankly, I'm puzzled and disturbed at the possibility which may emerge, not only in connection with the

KW!

Discussion

397.

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VORWALD COLL.
BOX 59

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Discussion

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Frankly, I am puzzled and disturbed at the possibility which may emerge, not only in connection with the question of an etiological relationship between cancer of the lung and asbestos, but also between cancer of the lung and other local irritants.

In 1949, I published a note of the deaths from asbestosis, asbestosis with tuberculosis, and either of these also with cancer of the lung, recorded in the United Kingdom from 1924 to 1947. I placed alongside them for comparison the corresponding data we have from silicosis and cancer of the lung. These were factual data which speak for themselves insofar as they go, but I attempted to make no deductions, since I felt the asbestosis deaths were too few for the purpose. I have now brought these figures up to date.

✓ The corresponding figures for silicosis deaths amount to several thousands. They were, I think, sufficiently large to show that there is no etiological relationship between silicosis and cancer of the lung and in this I/^{am} fortified by the data produced by the South African experts. I will not weary you, therefore, with the additions in the last three and a quarter years to our silicosis figures.

Briefly, therefore, our latest figures for recorded deaths from asbestosis, asbestosis with tuberculosis, or either complicated with cancer, are 306. I am excluding ten of these where there is also cancer of sites other than the lungs. Our net figures are, therefore, 296 deaths and of these forty-eight or 16.2 per cent were associated with primary cancer of the lung. This shows an increase from a 13.2 per cent disclosed by our earlier figures. This means that, as more deaths from asbestosis come to hand, the cumulative percentage of deaths with complicating cancer of the lung is rising rather than falling.

Now, where do we go to from here? Are these figures sufficient to indicate a casual relationship between the retention of asbestos dust in the lungs and subsequent cancer of the lung? The small numbers certainly dictate caution. Nevertheless, they represent the great majority of the deaths from asbestosis which have occurred in the United Kingdom during the past quarter of a century.

Nowadays, because of the greatly increased awareness of workers, doctors, and others in the neighborhood of asbestos plants, of the

disease; because of the duty of coroners to investigate with autopsy and to report to my department deaths suspected to be due to occupational disease, whatever it may be; because of the compensation benefits accruing to cases of pneumoconiosis; because of the system of factory inspection in force; because of our periodical check-up of the death certificates provided by the Registrar General and for other reasons, it is likely that very few deaths from asbestosis escape notice.

Therefore, with confidence, I think we can say that the figures point to the necessity for the collection of similar data from other communities with an asbestosis hazard, so that by means of statistical examination of the global figures so obtained we can settle the question of whether cancer of the lung is an additional risk of asbestos exposures.

I would remind you here that these figures include all the deaths from asbestosis, during the period in question, which we can trace and which have been verified pathologically. Undoubtedly, the deaths of some cases, which to my personal knowledge had developed asbestosis round about 1930, have escaped notice, for reasons about which we can only surmise: they may have emigrated or died from other causes, including primary or secondary lung cancers, and thus the asbestosis and occupational factors have passed unrecorded. For the reasons I have mentioned, however, such deaths have been increasingly unlikely to escape the net during the past 20 years.

KL-4

I mention this more particularly because of the real danger of duplication of cases in compilations of global figures from different countries. Often there have been reports in the United Kingdom of cases or of series of cases from different observers of particular aspects of the disease, and often the same cases have appeared in more than one of the reports. There are no duplicates in the figures given here.

✓
✓
✓
✓

There is another point to be borne in mind and that is that the population at risk from asbestos is small relatively to other industries and it fluctuates. A war brings a great increase of people at risk, because obviously there is a great demand for high class asbestos insulation for use in warships, and other types of work. Then when peace comes the demand drops off again, so that one can never really isolate a population, for instance, to which one could relate particular cases of asbestosis, assuming that asbestos is a factor in this matter.

We must be careful, therefore, as I have implied, that we do not accidentally magnify the risk. On the other hand, it is possible for a risk to be concealed because of an erroneous estimation of the population exposed to the risk. Thus, as I found a number of years ago in the coke-oven industry, only a small proportion of those employed are exposed to tar or tarry vapours, and in them the risk of cutaneous cancer is about six times that of the general population. Yet if one relates the cases to the whole population of the coke-oven trade, the risk is

entirely submerged. In other words, the risk is concealed in the overall numbers erroneously assumed to be at risk.

✓ And we shall have to consider this point in relation to asbestosis and cancer of the lung; a considerable proportion of workers in the asbestos industry have very little exposure to asbestos - on the other hand, we do not know whether much or little exposure to asbestos is required to trigger-off the cancer of the lung, if, in fact, there is such a relationship.

This morning Dr. Lynch asked, "What is the degree of asbestosis?" In fact, all these people have been autopsied and all of them died primarily of asbestosis, whether they had tubercle or cancer also.

I will not weary you with the statistical approaches we have made to our slender data, but will content myself with a rough breakdown of the figures.

Of the 296 deaths we are considering, 172 occurred in males and 124 in females. Eighty-two or 27.7 per cent, half males and half females, were associated with pulmonary tuberculosis. The mean age at death was 48.2 years, with a range of 24 to 73 years for the cases of asbestosis; 39.3 years with a range of 17 to 66 years for the cases of asbestosis with tuberculosis; and 53.4 years with the range of 32 to 77 years for the cases of asbestosis and cancer of the lung.

The mean duration of exposure was 15.7 years with a range of 1 to 48 years for deaths from asbestosis; 11.1 years with a range of 1 to 33 years for deaths from asbestosis with tuberculosis,

✓ and 19 years with a range of 2 to 42 years for deaths from asbestosis with cancer of the lung.

✓ The mean duration of life between cessation of exposure and death was 6.5 years with a range of 1 to 32 years for deaths from asbestosis, 4.7 years with ^a range of 1 to 21 years for deaths from asbestosis with tuberculosis, and 5.8 years with a range of 1 to 26 years for deaths from asbestosis and cancer of the lung. You see the ranges are very wide indeed, which is of some interest. Now, there are indications, from our data, that our preventive regulations which came into full operations in 1932, twenty years ago, are having an effect and this may be the explanation of why the incidence of complicating cancer of the lung appears to be rising.

✓ Today, we do not encounter cases of extreme massive fibrosis and gross bronchiectasis and even anasarca and a 'nutmeg' liver with death (uncomplicated with tuberculosis or cancer of the lung) at an early age, within 5 to 7 years from the commencement of exposure. Those cases do not seem to occur. The duration of exposure which results in death from asbestosis is lengthening, the age of death is increasing and the degree of fibrosis found at autopsy is less.

✓ This means that cases of asbestosis are living to the cancer-occurring age period, and if - and I say if - there is a relationship between asbestosis and cancer of the lung and if irritation, let us surmise from the asbestos fiber/ is a co-carcinogen with a long lag

KL-7
period, then we would expect an increased incidence of cancer of the lung over that found in the general population to become evident.

✓
Regarding these cancer cases, the mean age of death, as I said, was 53.4 years. and the range 32 to 77 years, and the mean duration of exposure 19 years with a range of 2 to 42 years. Now, the occupations involved may be interesting, but I would certainly not attempt to draw any deductions, since several occupations with different dust exposures were - and to a lesser extent nowadays are - carried out in the same room. Nevertheless 10 of the 48 cases were associated predominantly with weaving, 8 with disintegrating, mixing or opening fibre, 7 with pipe and boiler covering, and 6 with mattress making - all very dusty jobs in the earlier days.

✓
Well, what then is the etiological factor, if any, here? I would be most grateful for your views. I know that physical irritation as a direct carcinogenic agent has been rather discounted by the cancer experts, but I remember that a United States expert, Dr. Oppenheimer, has done some experimental work with cellophane wrapped round the kidney, which may be a pointer.

DR. RHOADS: Dr. Lynch, was there no correlation in your series between the extent of asbestosis and the occurrence of neoplastic change?

DR. LYNCH: The three cases of carcinoma in our series occurred in advanced cases of asbestosis, not in minor conditions where a diagnosis is made merely because asbestosis might be present.

DR. LANZA: I find it difficult to believe that the cancer incidence among asbestos workers in the United States could be anywhere near as high as it appears to be in England. We may have missed some cancer cases but I do not believe we could have missed so many if they had occurred to the same extent they apparently do in England. It is true, of course, that as soon as the harmful effects of asbestos were recognized by American industry a program of cleaning up the plants was instituted, and although all asbestos plants in the country have not attained perfection in industrial hygiene, great strides have been made, particularly in the plant in Charlotte, Dr. Lynch's city. With the number of workers in the industry so small, it is obvious that we shall never have the clinical experience with asbestosis that we have had with silicosis.

I used to hear frequently in Canada that the Canadian asbestos is less damaging than the kind used in England where the major part of the asbestos used, I am informed by my English friends, is Rhodesian asbestos, a variety quite dissimilar in structure and physical characteristics to the Canadian kind. I am told also that there are differences in the Canadian varieties, between the asbestos mined at the town of Asbestos and the kind mined not far away at Thetford. Since experimental studies in the past few years have shown that different types of silica may produce entirely dissimilar pathological effects, I am beginning to wonder whether we have given sufficient attention to differences in the type of asbestos in our attempt to explain variations in the rate of asbestosis.

KL-9

✓
✓

DR. SMITH: I had an opportunity to visit Dr. Gloyne shortly before his death and to learn something about the material he had assembled in England. He had 17 cases in which there was co-existent asbestosis and carcinoma of the lung. In his series there was no detectable relation between the degree of asbestosis and the presence or absence of cancer. His series came from a plant that used the Rhodesian blue asbestos which, as Dr. Lanza has pointed out, has certain characteristics that are different from those of Canadian asbestos. The Rhodesian variety has a more brittle fiber and, therefore, gives rise to more dust. I understand, however, that some of the British cases had been exposed to Canadian asbestos.

✓

Earlier the question of carcinogenicity^{of}/nickel was brought up. Recently, I visited Dr. Amor, formerly medical officer for the plant in Wales where the nasal sinus tumors had been observed, and Dr. Morgan, who is the medical officer there at present. They stated that the men who developed the nasal sinus tumors had not been employed in the part of the plant where there might be exposure to nickel carbonyl. Both doctors were very reluctant, therefore, to believe that nickel carbonyl was a factor in the nasal sinus tumors observed. They pointed out that the tumors which had occurred were in men exposed to arsenical dust from the calciners and that cancer of the nasal sinus was not seen in men who were employed in the industry only after 1924, when the calciners were re-designed.

DR. RHOADS: Dr. Merewether, would you comment on these remarks?

DR. MEREWETHER: I am afraid I cannot comment on all of them, but I think I can partially answer some and that Dr. Knox can answer others.

First of all, I think Dr. Lanza is misinformed about the proportions and kinds of asbestos used in England. The original cases of asbestosis, fifty years ago, were all from Canadian asbestos. The earliest commercial asbestos work in England was in the early seventies of the last century and there may have been some Italian fiber used here then, but very quickly the trade concentrated on the white Canadian chrysotile - namely a hydrous magnesium silicate.

✓
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✓
Later on, the Rhodesian chrysotile, the South African blue crocidolite, and another South African type, amosite, also mainly an iron silicate, came into use. Now, of all of these, amosite, the brown long-fibred material, is the most brittle and dusty - to such an extent that its use in England has declined. I do not believe they use any blue there at all and they have had a number of cases of cancer of the lung, as one might expect. Of course not all the cases of cancer of the lung, even if there is an association with asbestos, can be ascribed to it because there must have been an ordinary incidence of cancer of the lung irrespective of this possible factor.

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If Dr. Knox were here, he would be able to tell us about his plant, which is the largest in the country, about the types of asbestos they use, and about his experience of cancer of the lung. The fact is that mainly chrysotile is used in his factory.

Concerning the so-called nickel cancer, it is quite correct that none of us think that nickel carbonyl has anything to do with it as such.

Dr. Amor, Dr. Morgan, and myself feel that the re-building of the old calciners had a crucial effect on the risk, although there was some change in the constitution of the semi-refined matter shipped from Canada. The difference - as I know, because I saw them before and afterwards - between the old and extremely dust calciners and the modern practically dustless building is enormous.

DR. LEVIN: Dr. Knox informed me that in the plant under discussion there were from 1932 to 1951, exactly 16 cases of lung cancer. Unfortunately, Dr. Knox did not have sufficient data to enable him to say whether or not that was an excessive number of cases.

BY DOCTOR MEREWETHER:

Doctor Rhoads, Ladies and Gentlemen: I am greatly honored to be invited to attend this erudite meeting which has now, by virtue of the diverse and inspired work of my old friend, the late Doctor LeRoy Gardner, and Doctor Vorwald, become an international event.

Now, only a foolhardy criminal would speak after the most stimulating papers of this morning, but I'll endeavor to do my best, and I shall not detain you long.

All I have been able to do in the short time available is to bring up to date our mortality and pathological data on this matter and lay them before you in the confident hope that if there is an explanation of them, and there are deductions to be drawn from them, I shall obtain them during this conference.

Frankly, I'm puzzled and disturbed at the possibility which may emerge, not only in connection with the

question of an etiological relationship between cancer of the lung and asbestos, but also between cancer of the lung and other local illness.

In 1948, I made a note of the deaths from asbestosis, asbestosis with tuberculosis and either of these with also cancer of the lung, recorded in the United Kingdom from 1924 to 1947. I placed alongside them for comparison, the corresponding data we have from silicosis and cancer of the lung. These were factual data which speak for themselves insofar as they go, but I attempted to make deductions since I felt the asbestosis deaths were too few for the purpose. I have now brought these figures up to date.

The corresponding figures for silicosis deaths amount to several thousands. They were, I think, sufficiently large to show that there is no etiological relationship between silicosis and cancer of the lung and in this I'm fortified by the data produced by the South African experts. I will not weary you, therefore, with the additions in the last three and a quarter years to our silicosis figures.

Briefly, therefore, our latest figures are recorded deaths from asbestosis, asbestosis with tuberculosis, or either complicated with cancer of the lung, with cancer, are 306. I am excluding ten of these where there is also cancer of sites other than the lungs. Our net figures are,

therefore, 296 deaths and of these forty-eight or 16.2 percent were associated with cancer of the lung. This shows an increase from a 13.2 percent disclosed by our earlier figures. This means that, as more deaths from asbestosis come to hand, the cumulative percentage of deaths with complicating cancer of the lung is rising rather than falling.

Now, where do we go to from here? Are these figures sufficient to indicate a causal relationship between the retention of asbestos dust in the lungs and subsequent cancer of the lung? The small numbers certainly dictate caution. Nevertheless, they represent the great majority of the deaths from asbestosis which have occurred in the United Kingdom during the past quarter of a century.

Nowadays, because of the greatly increased awareness of workers, doctors and others in the neighborhood of asbestos plants, of the disease, because of the duty of Coroners to investigate with autopsy and to report to my department, deaths suspected to be due to occupational disease, whatever it may be, the compensation benefits accruing to cases of pneumoconiosis, the system of factory inspection, our periodical check-up of the death certificates provided by the Registrar General, and for other reasons, it is likely that very few deaths from asbestosis escape notice.

Therefore, with confidence, I think we can say that the figures point to the necessity for the collection of similar data from other communities with an asbestosis hazard, so that by means of statistical examination of the global figures so obtained, we can settle the question of whether cancer of the lung is an additional risk of asbest¹os exposures.

Now, I should mention here that the figures in United Kingdom, that these figures include all of them, because they come to me, so that we must not add up excess figures and - the X figures and Y figures and Z figures, and think that they're all additional cases of asbestosis, with carcinoma of the lung. One can add up, of course, different countries, but one must be very careful adding up or duplicating the same cases in the same countries.

There is another point that was brought up this morning, that the population risk of asbestos is small relatively to other industries and it fluctuates. A war brings a great increase of people, because obviously, there is a great demand for high class asbestos insulation for use in war ships and other types of work, and then it drops off again, so that one could never really get a population, for instance, to which one could relate a particular case of asbestosis, assuming that asbestos is a factor in this matter.

Now, another thing is the reverse which I think has got to be brought in mind, into mind, that when one is considering a hazard in an industry and one wants, as the statisticians always do want, something like a world population before they give us anything very satisfactory about the matter, that you - you say that, for all purposes, the population of an industry is say two hundred thousand, your very risk may be lost in the mass of the population.

That occurs, for instance, in coal carbon trade, where I tried a number of years ago, a relatively small portion of people exposed to tar or tarry vapors, and they're lost in the whole population of the trade, but when you limit them to the people who are exposed to tar and tarry vapors, you find there is six times the risk of cutaneous cancer. In other words, the risk was lost in the overall numbers assumed to be at risk.

And, it is something like that in the asbestos trade. A lot of people don't have much exposure to asbestos at all. Now, as far as the rate in asbestos, I haven't got into that yet, but following a remark made this morning, I will try and do so.

Then Doctor Lynch did say, what is the degree of asbestosis. Now, all these people have been autopsied and all of them died primarily of asbestosis, whether they had tubercle or cancer later.

Now, I won't weary you with the statistical approaches. Besides that, I'm very frightened of statisticians. They're going to die if I find an error in this. Not quite as bad as a psychiatrist, of course. I'm looking for the day when we have a statistical psychiatrist. Well, just another page of this, sir, then we'll be ready to throw me out.

Of the 296 deaths we are considering, 172 occurred in males and 126 in females. Eighty-two or 27.7 percent, half males and half females, were associated with pulmonary tuberculosis. The mean age of death was 48.2 years, with a range of 24 to 73 years for the cases of asbestosis. 39.3 with a range of 17 to 66 years, for the cases of asbestosis for 5r with tuberculosis and 53.4 years with the range of 32 to 77 for the cases of asbestosis and cancer of the lung.

The mean duration of exposure was 15.7 years with a range of one to forty-eight for deaths from asbestosis. Some took a Hell of a long time to die -- 11.1 with a range of 133 - no, I put the wrong one, I'm sorry, 11.1 years, with a range of 33 years with asbestosis with tuberculosis and 19 years with a range of 2 to 49, 2 to 42 years for asbestosis with cancer of the lung.

The mean duration of life between cessation of exposure and death was 6.5 years with a range of 1 to 32 for asbestosis, 417 years - that should be 4.7 years with

range of 1 to 21 for asbestosis with tuberculosis, and 5.8 years with a range of 1 to 26 for asbestosis and cancer of the lung.

You see the ranges are very wide indeed, which is of some interest. They say with tubercle, you can get tubercle any time. It can come on them at any time and kill them at any time. Now, there are indications, from our data, that our preventive regulations which came into full operations in 1932, twenty years ago, are having an effect and this may be the explanation of why the incidence of complicating cancer of the lung appears to be rising.

Today, we do not encounter cases of extreme massive fibrosis and gross bronchiectasis and even a sarcoma 'nutmeg' liver with death, and complicated with tuberculosis or cancer of the lung at an early age, within five to seven years from the commencement of exposure. Those cases don't seem to occur. The duration of exposure which results in death from asbestosis is lengthened, the age of death is increasing and the degree of fibrosis found at autopsy is less.

This means that cases of asbestosis are limited to the cancer occurring age period, and if - and I say if there is a relationship between asbestosis and cancer of the lung and if irritation, let us surmise from the asbestos fiber is a co-carcinogen with a long exposure period, then

we would expect an increased incidence of cancer of the lung over that found in the general population to become afterwards.

Regarding these cancer cases, the mean age of death, I said was 53.4 years, so I put this in for - since I found I'd be kicked off if I didn't. Now, the occupations involved are somewhat interesting. They're always different, as you know, because sometimes two occupations carried out in the same room with different dust exposures or with other different factors, but it is a little interesting to see that the majority of these forty-eight cases, that the more dusty the process, of course, it's most fallacious, but still we watch with care, for instance, weaving. Weaving is notoriously dusty, one case there with ten of these cancer lung cases, one case also associated with tarding and spinning that the man had done, and one case mixing and sorting, disintegrating and mixing, either - well, eight pipe and boiler coverers, including mixing; seven, and mattress making, six and all the rest are below that, and all those are dusty jobs.

Well, what then is the etiological factor, if any here? And, I'd be most grateful for your views. I know that physical irritation as a direct carcinogenic agent has been rather debunked by the cancer experts, but I remember that some recent work by a United States expert,

Doctor Oppenheimer, has - who has done some experimental work with cellophane wrapped around hinges, which may be a pointer to this factor if it is material. Thank you.

BY DOCTOR RHOADS:

Thank you, Doctor Merewether. Is there a comment about the Merewether discussion? (No response).

Doctor Lanza, I think you can make some comment on this.

BY DOCTOR LANZA:

Mr. Chairman, Ladies and Gentlemen, before going into this brief statement of mine on chromates, I would like to make a few comments on Doctor Merewether's observations.

I was very much impressed with what Doctor Merewether said, and there is no question but what a very careful, painstaking job has been done in Great Britain on the subject of trying to determine what the hazard might be in the asbestos industry with respect to cancer.

There are just a couple of comments, I'm not saying one way or the other, that it may or may not influence the incidence of cancer. I would raise a couple of points which I think all of us might keep in mind, and which might possibly have some bearing on the situation in England.

Now, I have visited, somewhat extensively, I think, nearly all the asbestos plants of the United States, and the mines and the mills in Canada and have spent quite a lot of

time there, and have spent a lot of time reviewing films, and not long ago, spent several days with Doctor Kenneth Smith on asbestos, reading his films.

I find it difficult to believe that our cancer incidence among asbestos workers could be anything like as large as it seems to be in England, because while I'm perfectly free to admit that we may have missed cancer cases which we should have not, on the other hand, I don't think we could have missed all of them if they had occurred, into the same extent that apparently they do in England.

Now, that is simply an observation of mine. Of course, it is true that as soon as the business of the harmful effects of asbestos once took hold in the American industry, they did start to clean up their plants and, while I wouldn't say that all the asbestos plants in the country were marvels of industrial hygiene perfection, nevertheless great strides have been made, and particularly, for instance, in the plant in Doctor Kenneth Lynch's town in Charlotte, South Carolina, where they have done a very beautiful job, so that with the small number of people in the industry, it's obvious that we are never going to have the clinical experience with asbestosis that we have, for instance, with silicosis, because there is not going to be the material to work on.

Now, back of that, there is still one more question.

That is the nature of the asbestos itself. Now, when I first used to go to Canada, I used to hear frequently that, up there, that the Canadian asbestos was less damaging than the kind they used in England, I believe, and I have put that comment down to what we might call the national or local patriotism, you know, like the rivalry between towns, so that you so frequently see, but the fact remains the same, that in this country, we do use the asbestos, mostly the asbestos that we get from Canada, and I believe, from what I hear from our English friends, that a great deal, if not the major part of the asbestos used in England, is Rhodesian asbestos, which is quite dissimilar in its construction and in its formation, its physical characteristics, to the kind that comes from Thetford and from asbestos in Canada.

Now, that may have nothing to do with it, and up until two or three years ago, I dismissed that. I thought that was just purile, but since we have in the last few years, learned that different types of silica, for instance, produce entirely distinct pathological effect, I am beginning to wonder if we have not failed to pay sufficient attention to the type of asbestos which varies even, as I am informed, there is a difference between the asbestos that is mined in the town of Asbestos and the kind that is mined not far away in Thetford, and yet both of these, in turn,

are quite different from the asbestos that come from Rhodesia, and possibly from other parts of the world with which I'm not acquainted.

And, having in mind the lesson we've learned in the silica dust, I think that is something perhaps that we should pay more attention to. Now, I simply raise that point, because I think it may have a great deal more significance than we have been inclined to give it.

Now, I have summarized - I have summarized my remarks here very briefly, because the topic of chrome is going to be handled by Doctor Anna Baetjer, who has done the experimental work in chrome, and has a more intimate knowledge of it from that point of view than I do.

The available evidence is that chromate workers do have an incidence of cancer of the lungs in excess of what might be expected. Beyond this, we do not as yet know a great deal.

Interest in chrome as a possible carcinogenic agent, with particular respect to lung cancer, is comparatively recent. The officials of one of the largest chrome producing companies in this country supplied the impetus that started interest in this subject in the United States, their apprehensions to tangible form, in setting on foot the statistical survey conducted by Machle and Gregorius, and published in the Public Health Reports on August 27th, 1948

Efforts were made to reduce exposure in chromium plants and more intensive medical service was inaugurated with a much more free use of X-ray examination, and I venture Doctor Baetjer will tell you of her work in Baltimore with experimental animals.

Now, Doctor Baetjer published a comprehensive paper on pulmonary carcinoma in chromate workers in the Archives of Industrial History in 1950, and up to now, there is not much more to say that was not said in her paper. In a word, as the saying goes, she covered the subject 'from soup to nuts' and as far as our question stated along that line, goes.

Now, fortunately, during 1952, the Division of Occupational Health of the United States Public Health Service, with the assistance and cooperation of the industry, conducted a survey of the whole chrome industry. Through the courtesy of Doctor Seward E. Miller, in charge of the Division of Occupational Health, I have been given a copy of the medical portion of their admirable report. Doctor Miller was kind enough to make that available to me when I asked him if he was going to present his findings here and he said no.

The report, however, is in the press now, and as it will be presently available, I will only refer to it in a very brief summary. I quote from the Public Health

Service Report: All specific causes of mortality in chrome workers, the chrome workers had frequency rates that were not greatly different from other workers. However, cancer with a rate of 7.1 compared with .7 stands out as markedly in excess for chromate workers. For cancer of all sites, the actual number of deaths of chromate workers was approximately four and a half times the number that would have been expected had the cancer rate for all males in the United States prevailed. When cancer of the respiratory system was observed separately for chromate workers, nearly twenty-nine times as many deaths as were expected were found. All other types of cancer failed to show an excess among chromate workers.

It is obvious -- that's the end of the quote -- it is obvious that much research and experimentation remain to be done before we can talk with more assurance of this situation. In the meantime, the industrial plants are being cleaned up and in some cases re-built with the hope and expectation of steadying this tide of cancer cases.

Before leaving this topic, I again would like to make a plea for the more careful and precise use and association of words. The evidence of the relationship of chrome exposure and pulmonary cancer appears conclusive. This cancer of the lungs in chrome workers does not follow, as far as I can ascertain, does not follow, nor is it dependent

upon a pre-existing pneumoconiosis. I mention this so that there may not be any false conclusions drawn from the association of these two terms as they are used in heading up our program. Thank you.

BY DOCTOR RHOADS:

With very characteristic precision, Doctor Lanza has laid to rest any illusions or questions that might be raised concerning the relation of chrome to pulmonary cancer. Is there a comment on Doctor Lanza's presentation?

BY DOCTOR RICHARDSON:

Who were the workers that were effected in this?

BY DOCTOR LANZA:

As far as I know now, I can't tell you positively until I have had a chance to digest the Public Health Service report, but you know these are small plants and in small plants, men shift around, so that not only do we not know what particular chrome substance or what particular process in the manufacture of chrome is responsible, if it is a cancer, but we do not know or you can't depend on this group of men in this department or that group of men in that department, unfortunately, we can't do it.

BY DOCTOR RICHARDS:

Well, could we say when does the exposure to chrome begin and when does it end in the process of the handling of chrome?

BY DOCTOR LANZA:

Well, you would have to then. We had, I think, the engineering portion of the Public Health Service report, which will give the details of the process, the dust studies, and so forth, at different stages all along the line, from the time they get their ore out until it is shipped out of the plant.

BY DOCTOR RICHARDS:

Well, then, is there any hazard after it is shipped out of the plant?

BY DOCTOR LANZA:

As far as I know, there has been no complaint from any source, but that again is something we don't know.

BY DOCTOR RHOADS:

Doctor Hueper has comment.

BY DOCTOR HUEPER:

You asked the question whether the material shipped out of the plant will become carcinogenic. There have been reports from Germany that zinc pigment workers have developed carcinogen. The zinc chromate, the yellow die with which we spray our airplanes and with which we spray the oil tanks and oil pipe lines, if you see those beautiful bright yellow colors in the landscape of our refineries, that's zinc chromate. We are quite a bit interested to find out whether the painters who sprayed the zinc

chromate on our airplanes during the last war have increased cancer incidence. We don't know that. They can't be traced, but it seems to be.

BY DOCTOR VICKERS:

In regard to the previous question, I might say that in the chrome industry, workers are exposed to chromate in one way or another all the way through the plant. Some of your workers are exposed to chromium, and some to hexavalent chromium all through the operation.

BY DOCTOR RHOADS:

You can't pin down any particular step or form of chromium?

BY DOCTOR VICKERS:

No, some workers will have exposure to chrome in some form or other, but there are mixed exposures in one form or another and definitely to chromium all the way through.

BY DOCTOR RHOADS:

Would you care to comment on the zinc chromate observation, Doctor Vickers?

BY DOCTOR VICKERS:

No.

BY DOCTOR RHOADS:

Is there further comment? Doctor Baetjer?

BY DOCTOR BAETJER:

In the literature, there have been reported,

already in the literature, a hundred and thirty-four cases that are already on record. Of that number of cases of lung cancer, there were a hundred and twenty-one of those are in the chromate producing industry; ten are in the chrome pigment industry, that is where they are manufacturing the zinc chromate pigments, and the varying, the lead chromate pigments, and only two have I been able to find reported in the literature, among people who use chromium.

We had one case in Baltimore in a chrome plater, but he was also exposed to many other substances, and there is one reported in the literature who sprayed chromium or sprayed with chromium paint. Whether it was spray painting or not was not stated. So that, altogether, of the one hundred and thirty-four cases, there are a hundred and thirty-two or thereabouts that are in the chrome producing or chrome pigment producing industry, and only two, so far, in the chrome users.

There are a few other points, if I may add a few points to this, if Doctor Lanza will forgive me for adding onto his paper. I don't know whether he got over the point to you that we have never had, interestingly enough, any cancer of the skin in chromate workers, nor have we ever had any cancer of the nasal sseptum, even though a large percentage of these workers have perforation of the nasal sseptum.

There are some figures that might interest you concerning the duration of the exposure of these people until they get cancer. May I show one slide? This might interest you to show the distribution of these cases by length of exposure. If you just focus your attention, because we don't have much time, on this column and the range, you will see that the average age is fifty-two years, and the range was from thirty-one to seventy-two of these cases that have been reported in the literature.

The duration of exposure is the interesting thing here, and it varies from four to forty-seven years. These men have been exposed to chromium, before they developed their cancer. The interval between initial exposure and the diagnosis of cancer was approximately the same, because once it is - about seventeen, and the range is four to forty-seven.

Then between the end of exposure and the diagnosis of cancer is also interesting. It shows that in some cases, the cancer may develop many years after the end of the exposure. The interval between diagnosis and death is, as in all cases of lung cancer, an average of a very short period of time.

Many of these cases which we have had and which we have studied, have come to our dispensary claiming that their symptoms, their severe symptoms, didn't begin until

two weeks before they came to the clinic, and they could then be diagnosed as having lung cancer. The clinical symptoms and the pathology are no different in the chromate workers, as far as we have been able to determine, than in lung cancer of other origins. I think those are the principal points.

BY DOCTOR RHOADS:

That raises the question, Doctor Baetjer, of asymptomatic on cancer which simply means no symptoms referable to the lungs. What brought these patients to the clinic, symptoms to the lung?

BY DOCTOR BAETJER:

No, some came with lung symptoms, pain in the chest. Some of the patients I have read, came with symptoms referable to the metastases.

BY DOCTOR RHOADS:

Is there further comment on this?

BY DOCTOR SMITH:

Doctor Lanza, again commented on Doctor Mewether's discussion, asbestosis, and then went on to the chromate situation. I wonder if I may call for a little more discussion of the asbestosis problem.

At the end of the morning session, a question was brought up as to association of lung cancer with the degree of asbestosis. Shortly before Doctor Glynn's death, I had

an opportunity to visit him and learn something of the material that he had assembled. He had seventeen cases in which there had been co-existent carcinoma and asbestosis in the lungs. In his series, there was no relation that could be detected between the degree of asbestosis and the presence or absence of cancer, or the degree of asbestosis and fight of cancer.

Now, his series came from a plant that used the Rhodesian blue asbestos which, as Doctor Lanza has pointed out, has certain characteristics that are different from the Canadian asbestos. It's a more brittle fiber and, therefore, gives rise to more dust. However, it was my understanding that some of the cases described in Great Britain, had had exposure to the Canadian white asbestos.

I wonder if any of our colleagues from overseas who are in the room, might care to comment on that?

I would like also, to make one further point in connection with a matter that both Doctor Devan and Doctor Rhoads brought out, and that is the experiment of nature, where one may vary circumstances and see if that has anything to do with a phenomenon that has been observed. The question of exposure to nickel, which was brought up this morning, is something that I have been asked about by several individuals over the lunch hour.

I recently had an opportunity to visit with Doctor

Amore who was formerly medical officer for the plant in Wales where these nasal sinus tumors had been observed, and I also visited Doctor Morgan who is the Medical Officer there at the present time.

They both stated that the man who has developed the nasal sinus tumors were not men who had been employed in the part of the plant where there might be exposure to nickel carbonel, and they were, for that reason, very reluctant to believe that the nickel carbonel was a factor in the nasal sinus tumors that had been observed.

They further pointed out that the tumors that had occurred were in men who have been exposed to the calciners, that is the ores with the - that since 1924, when the calciners were redesigned, they have not seen cancer of the nasal sinus in men whose employment in the industry has taken place only subsequent to 1924, so that it would seem that there is an instance which fulfills the stipulations that were raised this morning as to whether an experiment of nature might be performed which would have bearing on the question of an occupational hazard in the way of a respiratory tract carcinoma.

BY DOCTOR RHOADS:

Doctor Merewether, would you comment on these remarks?

BY DOCTOR MEREWETHER:

I couldn't comment on some of them, but I think I can partially answer some of these, sir, and I think Doctor Knox can answer conclusively one of them.

First of all, I think Doctor Lanza is misinformed about the proportions of asbestos used and where they are used in England. The original cases of asbestosis, fifty years ago, were all Canadian asbestos. The earliest asbestos work in England was either seventy-eight or eighty-two and there may have been some crystalline fiber then, but very quickly the asbestos trade concentrated on the white Canadian crystalline.

Later on, the Rhodesian asbestos and the Rhodesian - the South African blue, and the third one which is also an iron silica, the amacite, came in. Now, of all of these, the amacite is definitely the most brittle. It has short fibers, it's brown and it's got fibers as long as that (indicating) whereas the best Canadian spinning fibers have only got fibers as long as that (indicating) and the best blue is about that.

Well, there is no doubt about the dustiness of the amacite. That is the brown long fibered stuff, to such an extent that it's very little used today, so it's at the period where some of these cases are concerned, or where the cases of asbestosis occurred - some of my old friend Glynn's cases occurred - there was a good deal of blue and

BY DOCTOR RHODES:

Doctor Merewether, would you comment on these remarks? 419.

DOCTOR MEREWETHER:

I am afraid I cannot
~~I couldn't~~ comment on ~~some~~ ^{all} of them, but I think I can partially answer some of these, ~~etc.~~ ^{others.} and ~~I think~~ ^{that} Doctor Knox can answer ~~conclusively~~ ^{one} of them.

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Later on, the Rhodesian ^{chrysotile,} asbestos ~~and the Rhodesian~~ ^{crocidolite, another South African type,} ~~is~~ the South African blue, and the ~~third one which is~~ ^{into use.} ~~also an iron silicate,~~ ^{mainly silicate} the amacite, came in. Now, of all of these, the amacite is ^{amorphous, the brown long fibered material} ~~definitely~~ the most brittle, ^{and dusty - to} It has such an extent ~~that its use in England has declined.~~ short fibers, it's brown and it's got fibers as long as that (indicating) whereas the best Canadian spinning fibers have only got fibers as long as that (indicating) and the best blue is about that.

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a good deal of amacite used.

Now, I think Doctor Knox will be able to tell us, in his plant, which is the biggest in the country, what about the types of asbestos they use in that particular factory, and the - whether they have had any carcinoma of the lung.

Now, about the nickel cancer, it is perfectly correct that none of us think that nickel carbon has anything to do with it as such for the very simple reason that - well, one reason is that nickel carbon is so toxic that we should build a factory in order to get the - in order to get the little cancer, and of course, it might act in that proportion, then I should imagine a lot more people would have been effected.

Doctor Amore or Doctor Morgan and myself feel that the - that the re-building of the old calciners was the crucial change in the matter, though there was some operation in the constitution of the materials which are shipped from Fort Home in Canada. The difference, as I know, because I saw it before and afterwards, between the old and extremely dusty calciners and the modern build, is enormous. Therefore, there may be some hope in the fact that perhaps a lot of big dosage is necessary to produce some of these cancers.

BY DOCTOR RHODES:

I do not think they use any blue there at all and they have had a number of cases of cancer of the lung, as one might expect, of course not all the cases of cancer of the lung, even if there is an association with asbestos can be ascribed to it because there must have been an ordinary incidence of cancer of the lung irrespective of this possible factor. 420.

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about the types of asbestos they use in that particular factory, and the ~~whether~~ ^{about his experience of cancer} they have had any carcinoma of the lung. The fact is that mainly eriposite is used in his ~~plant~~ ^{factory}.

Concerning so-called ^{quartz} ~~nickel~~ cancer, it is perfectly correct that none of us think that nickel ~~carbon~~ ^{carbonyl} has anything to do with it, as such, for the very simple reason that - well, one reason is that nickel carbon is so toxic that we should build a factory in order to get the - in order to get the little cancer, and of course, it might act in that proportion, then I should imagine a lot more people would have been effected.

Amor,

Doctor ~~Amor~~ or Doctor Morgan and myself feel that the ~~that~~ the re-building of the old calciners ^{had} was a ^{effect on the use,} the crucial ~~change~~ ^{although} in the matter, ~~though~~ ^{semi-refined matte} there was some change operation in the constitution of the materials which are shipped from ~~Port Hope to~~ Canada. The difference, as I know, ^{them} because I saw ~~to~~ before and afterwards, ^{practically dustless} between the old and extremely dusty calciners and the modern building is enormous. Therefore, there may be some hope in the fact that perhaps a lot of big dosage is necessary to produce some of these cancers.

BY DOCTOR RHOADS:

Dr. Morgan and myself feel that the re-building of the calciners was the crucial change in the matter because there is an enormous difference between the old and extremely dusty calciners and the new buildings. Perhaps, to produce these cancers, a big dosage is necessary.

In that factory it is mainly chrysotile that is used - no blue asbestos is used, I believe - and there have been a number of cases of cancer of the lung, as one might expect. Of course not all cases of cancer of the lung, even if associated with asbestosis, should be included in the list, because there must have been an ordinary incidence of pulmonary cancer irrespective of any factor of asbestos.

DR. LEVIN: Dr. Knox informed me that in the plant under discussion there were, from 1932 to 1951, exactly 16 cases of lung cancer. Unfortunately, Dr. Knox did not have sufficient data to enable him to say whether or not that was an excessive number of cases.

Chapter Twenty - one

Pulmonary Cancer in Experimental Exposures to Beryllium

Arthur J. Vorwald, M.D.

The report I'm about to give.....

.....

.....cancer in human subjects.

Discussion

MR. MCCORMACK: What concentration of beryllium was used?

DR. VORWALD: The concentration was 0.01 milligrams of BeSO_4 per cubic foot of air.

DR. BAETJER: What dose was used for the intratracheal experiments?

DR. VORWALD: In the intratracheal experiments we injected into the rats 0.25 cc. of a 5 per cent suspension of the dust, once a week for 3 weeks. The total amount of dust injected was 37.5 mg.

DR. HARDY: In relation to life span, what age of a man would be comparable to the age of a 14 months old rat?

DR. VORWALD: I suppose that 14 months of exposure in the rat might be equivalent to perhaps 25 years of exposure in human beings. Our rats seldom live longer than two years, so believe that at 14 months they are approaching the age period when they may develop pulmonary cancer.

7, -
OK
By
W. J. ...
...
DR. MEREWETHER: The discussion has brought out the various aspects in this matter that must be considered. The important things are prevention and compensation for those unfortunate enough to get the disease and suitable nomenclature and definitions to ~~enable~~ ^{facilitate these aims.} these matters to be facilitated. Since the word pneumoconiosis has a sinister meaning to ~~some~~ ^{many} people, I deplore the use of the word, ~~except in medical circles,~~ to indicate something that does not cause disablement, sickness, or death.

The term pneumoconiosis is interpreted differently in different circles in relation to the circumstances or the purposes for which it is used;

~~To suit all people three types of definition of the term pneumoconiosis~~ must be considered. First, the widest conception of the word like that of the intelligent layman or general practitioner who merely means a dusty lung; second, the particular definition adopted some years ago by the ILO; and, third, a definition more narrow in type ^{as} like those used in different states and countries ^{for particular purposes.} In England pneumoconiosis is defined ^{for compensation purposes} at the present moment, as fibrosis of the lungs due to silica dust, asbestos dust, or other dust, and includes the condition known as "dust reticulation" ^{of the lungs} ~~of the lungs~~. Expert chest physicians understand the meaning of the definition and can apply it with justice. ~~Berylliosis, for instance, is at present being compensated as beryllium poisoning.~~

It is important to have for compensation purposes a definition which can be applied by specialists in the field. People that get a devastating disease should be compensated for it. It is unjust, however, by making a definition too wide, to compensate somebody who has not got the disease.

⁷ National Insurance (Industrial Injuries)

In our law, in the ~~Industrial Injuries~~ Act, there is a ^{unusual} provision for ^{prescribing} putting any disease ^{with retrograde effect} on the schedule as soon as it is shown to be occupational in character, and after it is on the list people who have ^{already} got the disease can be compensated for it. ~~The pneumoconiosis caused by bichromates is not a pneumoconiosis by our definition, so we may put it into the Act separately.~~

Mixed dusts, as everybody knows, produce very mixed types of X-ray appearances. You can have ~~mixed~~ dusts which are mixed in some areas of the country and not in others. ^{and yet they are given the same name. These materials sold as talc or French chalk, should contain no free silica or asbestos, yet may contain either or both with widely different effects on the lung.} Thus you can have a pure talc or so-called talc, ^(not by him) yet I have seen, through the courtesy of my old friend, that great man, the late Leroy Gardner, a slide of a lung which in the literature was labeled talc pneumoconiosis but which revealed rather obviously that the man died of silicosis and asbestosis together, whatever else the talc did.

Dust if inhaled ^{in sufficient quantity} may cause damage to the lung in spite of being classed as ~~active or~~ inert. But the main dusts that cause ^{identifiable pathology, and death} disability in disease we know, and if we can get a nomenclature suitable for them, we have achieved an objective which will help justice to be done to those ^{affected by them} known cases.

DR. VORNALD: We have had a discussion of disease - what is disease. Would you like to comment, Dr. McCann?

DR. McCANN: The interpretation of disease is a difficult question upon which to comment. As a clinician I think we have to relate the word disease to the occurrence of symptoms and we must realize that disease - in the sense that symptoms occur - may not be so extensive that it becomes a pathological process. A skilled athlete may develop a system of distress if the emotional distress is great enough, so we must consider the factor of great stress, the factor of normality or abnormality of the individual.

We must take into account the individual as a whole in regard to his ability to adapt himself to stress. He may have in one organ a pathological process for which he will compensate by the adapted processes in another organ so that under the same stress he may react differently than his neighbor. To define disease as distinct from a pathological process one must take those things into account, aside from cases like that of an individual with a definite pathological process produced by silica, who may never be put under stress sufficient to bring out symptoms. Therefore, from a clinician's standpoint, I believe one would have to say that disease is the difference in adaptation to stress, produced by a pathological process, to such a degree that the individual is unable to adapt with ease to the extent that a normal man would adapt himself.

DR. VORWALD: There is considerable agreement in our thoughts about definitions but there is some disagreement also. It is apparent that there are terms which need definition and clarification. In order that definite action be taken in this regard, it has been suggested that this Symposium invite Dr. Seward Miller, Chief of the Division of Industrial Hygiene of the U. S. Public Health Service, and Dr. Carl M. Peterson, Secretary of the Council on Industrial Health of the American Medical Association, to appoint a small committee of members representing organizations and individual experts concerned with the use of the word pneumoconiosis, for the purpose of considering the definition of the term with due regard to its application in the research, clinical, and legal fields and with the aim of establishing a common interpretation. Do you think such a committee should be formed and should it have corresponding members in England and South Africa and elsewhere, who might write their views to the committee?

DR. RICHARDS: I propose that your suggestion be made a motion and I second the motion.

DR. VORWALD: I think Dr. Seward Miller and Dr. Carl Peterson are in the audience and I shall place upon one or both of them the responsibility to arrange the committees. Will those in favor of the motion so indicate? (Response of ayes.) Those against it? (No response.)

DR. PETERSON: I'd like to see the American Public Health Association represented in the committee.

DR. GREENBURG: The American Public Health Association now has a committee at work on the terminology.

DR. VORWALD: Dr. Peterson's suggestion is a worth-while one and I am sure that the committee chairman will consider it.

Id Doctor Knox here? Do you care to comment on these remarks? (No response). Apparently not.

BY DOCTOR MEREWETHER:

Well, I can finish it if he won't.

BY DOCTOR RHOADS:

Why don't you?

BY DOCTOR MEREWETHER:

Because all of my information on this is derived from him; he's unduly modest. The fact is that it's mainly crysotiles used in that factory. I don't think they use any blue at all, and they've had a number of cases of cancer of the lung as one might expect. Of course, not all cases of cancer of the lung, even if it is due or associated with asbestosis, must be included in the list that we have, because there must have been an ordinary occurrence of cancer of the lung quite irrespective of any factor of asbestos.

BY DOCTOR LEVIN:

It just happens that I talked with Doctor Knox about his data at lunch, and he showed me the data which he has. That's the occurrence of cancer of the lung at his plant.

Now, from 1932 to 1951, they have had exactly sixteen cases of lung cancer. Unfortunately, Doctor Knox did not as yet have the information which would enable either him or anyone else to say whether that was an excessive

number of cases. That's the kind of statistics, which we presented to statisticians, that makes them wish that they did have a psychiatrist.

BY DOCTOR RHOADS:

Very proper statement, I'm sure. Is there further comment on our questions of chromate or nickel?

(No response).

We have dismissed these conclusive topics. We will proceed to the paper by G. Burroughs Mider, Director of Cancer Research, National Cancer Institute. Doctor Mider - on Experimental Pulmonary Cancer.

BY DOCTOR MIDER:

(Doctor Mider read a prepared paper, which is on file with the Saranac Laboratory).

BY DOCTOR RHOADS:

Certainly a very comprehensive dissertation on this subject. It is most unfortunate to suspend these experiments at this stage of medical science. I am most interested to hear advocated the correlation of clinical observation and epidemiological studies. May I ask you the reference to the ^{cerium} serial work, which is new to me, I think others might be interested in that since it hasn't appeared yet.

BY DOCTOR MIDER:

It has appeared in Lithgow & Finkel, Observations

Chapter Nineteen

Experimental Approach to the Problem of Pulmonary Cancer

G. Burroughs Mider, M.D.

Paper on file
The common occurrence.....

.....

.....of the lung in man.

EXPERIMENTAL APPROACH TO THE PROBLEM OF
PULMONARY CANCER

by

G. BURROUGHS MIDER

From the National Cancer Institute, National
Institutes of Health, Bethesda 14, Maryland

The common occurrence of primary tumors of the lungs in mice was recognized at least 20 years before pulmonary cancer assumed general clinical importance. Interest in these peculiar lesions has provided us with a mass of data which we can only summarize briefly today. The detailed studies of inheritance of cancer prosecuted by Dr. Maude Slye suggested to her that cancers in general were inherited as recessive Mendelian characters⁽³²⁾. This interpretation is open to considerable question. We mention it here because it represents almost our total knowledge of the heredity of spontaneous neoplasms in an essentially hybrid population -- and man is hybrid.

The development of inbred strains of mice by mating brother to sister for at least 40 generations has provided experimentalists with relatively homozygous subjects in which the specific anatomical types of cancer, the frequency with which they occur, and the age at which they appear are highly standardized. Results obtained by experimentation with these inbred mice are reproducible.

Study of inheritance of pulmonary tumor in inbred stocks resulted in the concept that the lesion was inherited as a Mendelian dominant

trait^(4,24). Consideration of the spontaneous appearance of these lesions, however, reveals that the incidence varies from less than 1 percent to practically 100 percent. Since there are so many strains with intermediate incidence, it would be extremely difficult to ascribe the inheritance of lung tumor or the susceptibility to pulmonary tumor as a single factor in inheritance^(1,10). The gradation, as Heston has pointed out, tends to prohibit the use of the terms dominant and recessive in respect to tumor inheritance. It now seems established beyond reasonable doubt that the inheritance of pulmonary tumors, and probably other forms of cancer as well, involves more than one gene. Geneticists have demonstrated linkage between multiple factor quantitative traits and single factor qualitative traits. Heston has shown an association between various quantitative genes of the mouse and susceptibility to pulmonary tumors. Susceptibility has been demonstrated to be associated with the linked genes waved-2 and shaker-2, the flexed tail gene, the hairless gene, and the lethal yellow gene (9,11,12,14). The last is particularly interesting since yellow mice, which are prone to the development of pulmonary tumors, are considerably fatter than their more resistant brown siblings. Confirmation of these data has been provided recently by Morgan⁽²⁵⁾. The other genes reduce both the weight and incidence of pulmonary neoplasms.

The ability of certain strains of mice -- notably Strain A developed by Strong⁽³⁴⁾-- to form a high proportion of pulmonary tumors has afforded an extremely sensitive tool for the quantitative estimation of

carcinogenic potency of some chemical compounds⁽³⁾. A number of apparently unrelated chemicals influence the genesis of such lung tumors in Strain A mice. Their influence can be measured in three ways. The lung tumors occur at an earlier age than would be expected, they affect a higher proportion of the population studied, and the absolute number of tumors formed is greater when a known carcinogenic stimulus is provided. The same compounds which influence the development of mouse pulmonary tumors are capable also of producing some cancers de novo.

Benzpyrene, dibenzanthracene and methylcholanthrene, for instance, elicit epidermoid carcinomas when painted on the skin, sarcoma when injected in subcutaneous tissues, and, in some cases, even tumors of the alimentary canal when fed in an appropriate vehicle. These compounds can elicit certain neoplasms, other than pulmonary, to which the strain may be spontaneously susceptible, such as mammary cancer or leukemia. The precise mechanisms by which these effects are mediated are not known at the present time, though some evidence suggests that the influence on the genesis of pulmonary tumors involves direct action of the carcinogenic agent on lung tissue as the potent agent circulates through the blood stream⁽²¹⁾.

Ethyl carbamate (urethane) is another chemical with potent effect on the mouse's lung tissue⁽²⁷⁾. It can be shown to increase the incidence and number of pulmonary tumors and to cause them to appear at an earlier age. In fact, the compound may exert its influence on the fetus in utero, apparently being transmitted through placenta ^(17,18). It would be impossible to detail the experience with all of the known chemical carcinogenic agents that have been tested for their effects on inducing tumors

of the lung, but there are some agents which do not seem to influence the occurrence of these peculiar neoplasms. No filtrable viruses have yet been incriminated in the etiology of pulmonary cancer. Gamma radiation enhances the development of tumors of the lungs in mice (20).

Spontaneous and induced tumors of the mouse's lung appear almost identical. Shimkin studied the action of methylcholanthrene on the lungs of 7 different strains of mice with varying incidences of spontaneous pulmonary neoplasms (31). He found no morphological differences among:

1. Spontaneous and induced tumors
2. Tumors induced in the 7 strains of mice studied
3. Tumors induced by methylcholanthrene and those produced by 1,2,5,6-dibenzanthracene
4. Tumors induced by subcutaneous, intravenous or intratracheal injection
5. Tumors induced by carcinogen dissolved in lard, in horse serum, or in cholesterol (pellet) or adsorbed on charcoal.

The ability of pulmonary tissues to react to a carcinogenic stimulus is a function both of the genetic constitution of the subject and the intensity of the stimulus. Heston and Dunn (13) recently transplanted pieces of lung successfully from mice of a strain resistant and of a strain susceptible to the induction of pulmonary neoplasms to hybrid mice, a cross between these two strains. Those grafts donated by the susceptible subjects

developed many more characteristic neoplasms in response to a carcinogenic stimulus than did the grafts from resistant donors. It would seem, then, that the genetic influence in large measure predestines the reaction of this particular tissue to its environment and that the susceptibility resides in the tissue itself. Comparable data have been published by Shapiro and Kirschbaum (30).

It is probably desirable to describe these tumors of the mouse's lungs. They may occur as single or multiple roughly spherical nodules visible through the pleura as circumscribed gray masses of granular tissue often producing a convex distortion of the pleural surface. The tumors affect both sexes equally. Histologically they appear as convoluted columns of cuboidal or columnar cells supported by a delicate, poorly vascularized stroma. They appear to grow expansilely and project into surrounding alveoli, which they ultimately fill. Metastases are not common but when present involve the mediastinal nodes and sometimes distant sites (36). Tumors are usually measured in millimeters but occasionally larger samples are found. Grady and Stewart ascribed their histogenesis to cells lining pulmonary alveoli (8). Orr dissents from this view and believes that they are of bronchiolar derivation (28). While the burden of evidence tends to support Drs. Grady and Stewart, we can conclude, at least, that this particular type of tumor does not arise from the bronchi or their major ramifications.

The lesions we have described are most certainly neoplastic but their precise classification is difficult. A tendency to call them

adenomas is increasing. One may make a reasonably good case for their cancerous character if structural changes alone are considered. Examination of their biological properties, however, confuses the picture for they seldom threaten life. The absence of metastasis is not an adequate reason to deny malignant properties in mouse neoplasms for unquestionably cancerous lesions often remain localized in this species. It seems probable that the spontaneous or induced pulmonary tumors of the mouse represent a spectrum of biological types among which locally invasive neoplasms predominate.

One of the most interesting phenomena that has been described in lung tumors of this kind is their behavior on serial transplantation to mice of the same inbred strain, which they invariably kill⁽²⁾. The sample transplanted is necessarily small, but over a course of five to ten successful transplantations the morphology of these masses undergoes a dramatic change, with sufficient frequency to suggest that it is not accidental. The cells lose their regular rectilinear appearance and assume various bizarre forms, ultimately becoming long and spindle shaped. They then present the general picture usually associated with sarcoma^(23,33). Whether this represents an actual change in the epithelial cells which appear to be the neoplastic elements, or some change in the stroma, is conjectural. The phenomenon can be observed in spontaneous tumors in which no chemical agent can be incriminated or in tumors induced with polycyclic hydrocarbons. In the latter instance, however, it seems clear

that the action is not mediated through continued contamination with an active carcinogenic agent which might produce sarcoma through acting on the host's own tissues.

It is curious, with the wide varieties of carcinogenic agents which will affect the spontaneous incidence of pulmonary tumor in mice, that experiments with tobacco have been completely ineffective. Flory has shown that tars which can be isolated from tobaccos have some tumorigenic potency when painted on the skin of mice⁽⁷⁾, but Lorenz and Stewart were unable to affect the incidence of pulmonary tumors in Strain A mice when they were exposed for 4 hours a day for 1 year to an atmosphere heavy with tobacco smoke, even though a residue probably derived from tobacco could be identified in the lungs on histological examination⁽²²⁾.

Campbell of the National Institute for Medical Research (London) reported his experience with mice exposed to inhalation of various dusts for 6 hours on 5 days of each week over a period of 1 year⁽⁶⁾. He divided his results according to the statistical significance of the difference between tumor incidence in exposed and unexposed subjects. He found that significance of increase was high in mice exposed to tarred road dust, similar dust in the presence of carbon monoxide, and a dust mixture containing equal parts of silica, iron oxide (Fe_2O_3), alumina and calcium carbonate. He considered also that samples of iron oxide, of Czechoslovakian pitchblende dust and tarred road dust that had been extracted with benzene also increased the pulmonary tumor incidence significantly.

Perhaps the most interesting finding was a lack of correlation between pulmonary dust deposits and tumors of the lung. In this sense, at least, the mouse responded as does man. A wider variety of histological types of pulmonary cancer was claimed among mice exposed to dusts than in the control group. Coal dusts produced no appreciable effect on the incidence of pulmonary neoplasms in the mouse.

Most species of common laboratory animals other than the mouse seem to have an excessively low incidence of spontaneous pulmonary neoplasms. Such lesions occur so rarely in the rat that Horn and Stewart were able to find very few reports of them recorded in the literature⁽¹⁵⁾. Urethane produces pulmonary tumors in albino rats similar to those which occur spontaneously in mice^(16,26).

It must be clear by this time that the spontaneous tumor of the mouse's lung is not precisely comparable to the great majority of pulmonary cancers that affect man. If we can accept the epidemiological studies of bronchogenic carcinoma -- and we use this term as synonymous with pulmonary cancer -- then we are confronted with a situation in which the apparent increase in pulmonary cancer during the past 25 years has affected males predominantly. Furthermore, almost all of the increase is probably due to epidermoid or undifferentiated carcinoma. Unfortunately the experimental production of bronchogenic carcinoma of the lung in any species must be quite difficult. Such a lesion was found by Orr and Bielschowsky following the administration of 2-acetylaminofluorine⁽²⁹⁾.

Their interpretation of the material is questioned by some workers. It is possible to produce squamous metaplasia of the bronchial epithelium in various experimental animals but, nevertheless, most of them prove resistant to development of a cancer which might have arisen in a metaplastic area. Lisco and Finkel found neoplasms in the lungs of rats that had been exposed to an aerosol containing radioactive cerium (cerium oxide)⁽¹⁹⁾. The work has been published only in abstract form but the investigators suggest that the cancers observed in their material originated in foci of metaplastic bronchial epithelium. A more detailed report of this work is urgently needed. I do not wish to anticipate the presentation of our host, Dr. Vorwald, but it would seem possible to induce unequivocally malignant neoplasms in the lungs of rats, a species with extremely low incidence of spontaneous pulmonary tumor, by exposure to contaminated atmospheres. Most of the work cited of studies on mice has used percutaneous or parenteral routes of administration for the carcinogens. Whether the use of inbred strains of mice is indicated in inhalation experiments seems conjectural. The combined effects of an agent inducing a particular form of tumor in which we are not greatly interested and the toxic side reactions that may be expected to accompany the administration of noxious substances may militate against our ability to produce the bronchogenic lesions that we wish to study. It might be preferable to use mice that are not susceptible to the development of spontaneous neoplasms of the lung. More extensive use of the inhalation technique seems to be indicated, particularly

since this seems to be the most logical route by which carcinogenic agents would have access to the human lower respiratory tract. I say 'most logical' because we know that highly potent chemicals may produce their cancerous effects at considerable distance from the site of administration. One of these, Betanaphthylamine, which produces cancer of the bladder in man, has to be metabolized to a 2-amino-1-naphthol compound before it has any carcinogenic effect⁽⁵⁾. We can not eliminate the possibility that even an ingested material may affect importantly the incidence of human pulmonary cancer.

If the inhalation route seems preferable, then our experiments must be designed to test the potential cancerous hazards that have been incriminated in the genesis of human cancer of the lung. Further experimental work on products of combustion of tobacco is greatly to be desired but one must consider seriously whether or not exposure to an atmosphere of tobacco smoke really reproduces the same situation as cigarette smoking in man. It is felt that the smoking of cigar or pipe does not constitute the same hazard as does the cigarette. Whether or not combustion products of these three types of tobacco are similar or identical should be established. It has been suggested that arsenic used to spray the growing plant may be responsible for the apparent association of excessive cigarette smoking and pulmonary cancer. This is a tenuous argument. Is it customary to spray the plants from which cigarette tobacco comes with arsenic and not to spray the tobacco that is used for cigars or pipes? If arsenic

were of any major significance in this phenomenon, one would expect to find some unequivocal manifestations of arsenical poisoning such as palmar and plantar keratoses, other cutaneous eruptions, and perhaps nasal septal perforations. Yet, no records of such paracanceroses have been recorded.

Evidence is beginning to accumulate that the incidence of pulmonary cancer in city dwellers is greater than that in residents of rural communities. I know nothing about the smoking habits of these two groups, but certainly contamination of the atmosphere in our industrial areas is a matter of common knowledge. Waller has recently isolated 3,4-benzpyrene from the smog of British cities⁽³⁵⁾. This is the known active carcinogenic agent found in soot and in coal tar. It enhances the incidence of pulmonary tumors in mice. The combustion of fuels may conceivably give rise to carcinogenic agents other than those already known. Data on the exposure of experimental animals to atmospheres similarly contaminated would be highly desirable.

There are a few industries in which there seems to be an inordinate amount of pulmonary cancer. These experiences offer a means of testing the susceptibility of experimental animals to the development of cancer of the lung with materials that almost certainly can produce the disease in man. I say 'materials' because, to the best of my knowledge, no chemically definable carcinogenic compound has yet been isolated from the mixtures that have been incriminated. Identification of such compounds

is a slow, tedious process which nevertheless should be pursued vigorously, not only from the standpoint of understanding better our problem of lung cancer in man but in the development of laboratory studies which will expedite our investigations. We all can agree that the acquisition of factual knowledge is a desirable end in itself but, when confronted with an important practical problem, our efforts should be directed towards its solution. Hence all facets of the enigma, the etiology of bronchogenic carcinoma in man, are pertinent to the discussion of laboratory experiments designed to study cancer of the lungs.

The evidence currently available does not seem to account for all of the excess of malignant pulmonary neoplasms that has been alleged to occur. The apparent relationship between cigarette smoking and cancer should be better defined by a study of large groups of smokers, a forward-looking project. We are told that a significantly high proportion of patients with cancerous lungs have a long history of rather extensive cigarette smoking but we need information on the relative frequency with which non-smokers and smokers of different types and degrees of tobacco consumption develop pulmonary carcinomas. Possibly the ladies may provide the answer in due course. If the increase has affected predominantly the masculine persuasion as seems indicated, we should expect some upward trend in the incidence of bronchogenic cancer among females in the foreseeable future. The ladies are smoking. That much is certain.

The preponderance of males in the population with pulmonary neoplasms may suggest that men are subjected to some cancerous hazard which women escape. This is strengthened by genetic studies of tumor development in laboratory mice. It seems unlikely that hereditary susceptibility to those murine tumors with which we are familiar is a sex-linked character in the genetic sense. Mammary tumors occur exclusively in female mice as spontaneous growths. They may be induced readily by estrogens among males of those strains in which the female is spontaneously susceptible. Occupational exposure might afford a reasonable solution to the problem if more and better evidence that such is the case could be obtained. This is an extremely difficult matter to evaluate. The historical approach of an adequate occupational history is far from ideal. It presupposes that the individual interrogated remembers the details of his employment accurately and is familiar to some extent with industrial processes. Frequent changes in job or in its details even within one plant are difficult to retain over the years, especially when the patient's mental acumen is blunted by his illness. A forward-looking program should be designed that is based on a common group study -- common employer, common trade. In any case the intimate and continuing interest and cooperation of industry is essential to success whether the approach be historical or futuristic. There is no real reason why such a study should be limited to cancer of the lung.

Experimental cancer research has amply confirmed the basic observations of Mr. Percival Pott and Sir James Earle that extrinsic agents can

cause cancer and not all subjects exposed to the noxious stimulus need develop a neoplasm. The study of cancerigenesis in animals has added other important concepts and facts. Susceptibility and resistance to cancer are determined by genetic influences. Susceptibility and resistance are purely relative terms for an overwhelming stimulus may overcome resistance. The carcinogenic stimulus requires a long time to produce its result in which effective dose rather than continuous exposure is a major determining factor. Non-carcinogenic agents and, indeed, naturally occurring substances important to the body's economy may enhance or inhibit carcinogenesis. A potent carcinogenic stimulus not only increases the incidence of specific anatomical types of cancer in a species but may cause them to occur at an earlier age than would the same specific kind of neoplasm in the usual course of events. The last statement is extremely important for our purposes. Perhaps we might arrive at a better conception of those factors that are especially important in the causation of bronchogenic carcinoma if we studied more intensively its victims less than 50 years of age. Experimental cancer research has provided one new clue for improving our clinical research attack on the etiology of cancer: relatively large doses of a carcinogenic agent stimulate the carcinogenic process and evoke tumors at a comparatively early age. The younger patients with pulmonary cancer should represent those who have either the greatest susceptibility or who have received a relatively intense stimulus.

The general tenor of this talk may lead one to feel that your speaker has unbounded confidence in the ability of the experimentalist to solve the clinical problem of etiology of bronchogenic carcinoma. His enthusiasm is tempered by realization that extrapolation from laboratory to clinic is fraught with many pitfalls. An azo dye no longer used to adulterate foodstuffs produces cancer in the rat's liver. It was consumed in considerable quantities by many of us who ate colored oleo-margarine during the first World War but no appreciable increase in the incidence or relative frequency of hepatic neoplasms ensued in the populace. Conversely the production of cancer in man by long continued ingestion of compounds containing trivalent arsenic is documented in such a way as to remove any reasonable doubt that cause and effect obtain. Nevertheless, no one has reproduced this situation in an experimental animal though many have tried. Nothing resembling Koch's postulates can be formulated today for cancers by which one may prove cause and effect.

One may conclude that the study of spontaneous pulmonary tumors in mice has been of little immediate practical value. This is a dismal view. The workers who labor so diligently over this vexing material have already taught us a lot. They have shown that general principles may be derived from investigating different types of cancer. They have pointed out the multiplicity of cancer-producing agents and defined how the effects produced may be modified. Practical applications of their knowledge depends upon the closest possible cooperation between experimentalists and clinicians. Only then will we accumulate the enormous stock of factual information that will point out the etiology of cancer of the lung in man.

SELECTED REFERENCES

- (1) ANDERVONT, H. B.: Susceptibility of mice to spontaneous, induced and transplantable tumors. Pub. Health Rep. 53: 1647-1665, 1938.
- (2) ANDERVONT, H. B.: Pulmonary tumors in mice. III. The serial transmission of lung tumors occurring in inbred mice. Pub. Health Rep. 54: 1519-1524, 1939.
- (3) ANDERVONT, H. B., and SHIMKIN, M. B.: Biological testing of carcinogens. II. Pulmonary-tumor-induction technique. J. Nat. Cancer Inst. 1: 225-239, 1940.
- (4) BITTNER, J. J.: Breast and lung carcinoma in A Stock mice. Pub. Health Rep. 54: 380-392, 1939.
- (5) BONSER, G. M., CLAYSON, D. B., and JOLL, J. W.: An experimental inquiry into the causes of industrial bladder cancer. Lancet 2: 286-288, 1951.
- (6) CAMPBELL, J. A.: Lung tumors in mice and man. Brit. Med. J. 1: 179-183, 1943.
- (7) FLORY, C. M.: Production of tumors by tobacco tars. Cancer Res. 1: 262-276, Apr. 1941.
- (8) GRADY, H. G., and STEWART, H. L.: Histogenesis of induced pulmonary tumors in strain A mice. Am. J. Path. 16: 417-432, 1940.
- (9) HESTON, W. E.: Relationship between the lethal yellow (A^Y) genes of the mouse and susceptibility to induced pulmonary tumors. J. Nat. Cancer Inst. 3: 303-308, 1942.

- (10) HESTON, W. E.: Role of heredity in tumor development.
J. Nat. Cancer Inst. 5: 161-171, 1944.
- (11) HESTON, W. E., and DERINGER, M. K.: Relationship between the lethal yellow (A^Y) gene of the mouse and susceptibility to spontaneous pulmonary tumors. J. Nat. Cancer Inst. 7: 463-465, 1947.
- (12) HESTON, W. E., and DERINGER, M. K.: Relationship between the hairless gene and susceptibility to induced pulmonary tumors in mice. J. Nat. Cancer Inst. 10: 119-124, 1949.
- (13) HESTON, W. E., and DUNN, T. B.: Tumor development in susceptible strain A and resistant strain L lung transplants in LA F_1 hosts. J. Nat. Cancer Inst. 11: 1057-1071, 1951.
- (14) HESTON, W. E., DERINGER, M. K., HUGHES, I. R., and CORNFIELD, J.: .
Interrelation of specific genes, body weight and development of tumors in mice. J. Nat. Cancer Inst. 12: 1141-1157, 1952.
- (15) HORN, H. A., and STEWART, H. L.: Spontaneous pulmonary tumor in the rat; report of a lesion. J. Nat. Cancer Inst. 12: 743-749, 1952.
- (16) JAFFEE, W. G.: Carcinogenic action of ethylurethane on rats.
Cancer Res. 7: 111-112, 1947.
- (17) KLEIN, M.: The transplacental effect of urethane on lung tumorigenesis in mice. J. Nat. Cancer Inst. 12: 1003-1010, 1952.

- (18) LARSEN, C. D.: Pulmonary-tumor induction by transplacental exposure to urethane. J. Nat. Cancer Inst. 8: 63-70, 1947.
- (19) LISCO, H., and FINKEL, M. P.: Observations on lung pathology following the inhalation of radioactive cerium. Abstract: Federation Proc. 8: 360, 1949.
- (20) LORENZ, E., HESTON, W. E., DERINGER, M. K., and ESCHENBRENNER, A. B.: Increase in incidence of lung tumors in strain A mice following long-continued irradiation with gamma rays. J. Nat. Cancer Inst. 6: 349-353, 1946.
- (21) LORENZ, E., and SHIMKIN, M. B.: Disappearance of intravenously injected methylcholanthrene in mice of different susceptibility to pulmonary tumors. J. Nat. Cancer Inst. 2: 491-498, 1942.
- (22) LORENZ, E., STEWART, H. L., DANIEL, J. H., and NELSON, C. V.: The effects of breathing tobacco smoke on strain A mice. Abstract: Cancer Res. 3: 123, 1943.
- (23) LUDFORD, R. J., and BARLOW, H.: Sarcomatous transformation of the stroma of mammary carcinomas that stimulated fibroblastic growth in vitro. Cancer Res. 5: 257-264, 1945.
- (24) LYNCH, C. J.: Influence of heredity and environment upon number of tumor nodules occurring in lungs of mice. Proc. Soc. Exper. Biol. & Med. 43: 186-189, 1940.

- (25) MORGAN, W. C.: The relation of the lethal yellow (A^Y) gene to pulmonary tumor formation and obesity in an inbred strain of mice. J. Nat. Cancer Inst. 11: 263-268, 1950.
- (26) MOSTOFI, F. K., and LARSEN, C. D.: Pulmonary lesions induced in Wistar rats by urethane. Am. J. Path. 25: 807-808, 1949.
- (27) NETTLESHIP, A., and HENSHAW, P. S.: Induction of pulmonary tumors in mice with ethyl carbamate (urethane). J. Nat. Cancer Inst. 4: 309-319, 1943.
- (28) ORR, J. W.: Histology and histogenesis of pulmonary adenomata of mice. Brit. J. Cancer 1: 316-322, 1947.
- (29) ORR, J. W., and BIELSCHOWSKY, F.: Histology of pulmonary tumors of rat induced by 2-acetylaminofluorene. Brit. J. Cancer 1: 396-400, 1947.
- (30) SHAPIRO, J. R., and KIRSCHBAUM, A.: Intrinsic tissue response to induction of pulmonary tumors. Cancer Res. 11: 644-647, 1951.
- (31) SHIMKIN, M. B.: Induced pulmonary tumors in mice. I. Susceptibility of seven strains of mice to the action of intravenously injected methylcholanthrene. Arch. Path. 29: 229-238, 1940.
- (32) SLYE, MAUDE: Genetics of cancer and its localization. Some fundamental aspects of the cancer problem. Acc. Pub. of the A.A.A.S. No. 4, pp. 3-16, The Science Press, Lancaster, Pa., 1937.

- (33) STEWART, H. L., GRADY, H. G., and ANDERVONT, H. B.:
Development of sarcoma at site of serial transplanta-
tion of pulmonary tumors in inbred mice. J. Nat.
Cancer Inst. 7: 207-225, 1947.
- (34) STRONG, L. C.: The establishment of the "A" strain of
inbred mice. J. Heredity 27: 21-24, 1936.
- (35) WALLER, R. E.: The benzpyrene content of town air. Brit.
J. Cancer 6: 8-21, 1952.
- (36) WELLS, H. G., SLYE, M., and HOLMES, H. F.: The occurrence
and pathology of spontaneous carcinoma of the lung in
mice. Cancer Res. 1: 259-261, 1941.

on Lung Pathology Following the Inhalation of Radioactive
^{Serium}
Serials, Abstract of Federation Proceedings, Page 360, 1949.

BY DOCTOR RHOADS:

Thank you very much indeed. I know many would be interested in this reference. I might also point out again in defense of the tobacco studies, that there has been some increase in cancer among women. Furthermore, much of the known cancer of women, may be adino-carcinoma, compared to carcinogenic.

Now, you're used to the term alleged increase, in man as not seriously meant. Do we not agree that the increase is real?

BY DOCTOR MIDER:

I think you and I can, Doctor Rhoads; I think Doctor Levin will join us, but there are still some die-hards who will not.

BY DOCTOR RHOADS:

This very interesting paper is open for discussion now.

BY DOCTOR FRIEDMAN:

I would like to ask Doctor Mider a question. You referred to the effect of coal dust on the incidence of cancer and you made the statement, I believe, that you did not think it had any effect, particular effect, upon the incidence of cancer. Now, we have seen -- or something to

that effect. Will you re-state - will you re-state your remark on that, Doctor Mider?

BY DOCTOR MIDER:

I don't know if I can; all I said was that the mere - or what I meant to say was that the mere presence of large quantities of coal dust in the lungs of miners over the years has apparently not been associated with any considerable frequency of pulmonary cancer.

BY DOCTOR FRIEDMAN:

That's the point. Well, now, I wanted to ask you a question. Do you know whether or not it actually has established the inhibition or has prevented the incidence of cancer in these individuals, because in our own group, we have been impressed with the fact that we have seen very few cases of pulmonary carcinoma in coal miners, and with respect to the incidence of the disease in the rest of our hospital population.

BY DOCTOR MIDER:

Well, come to think of it, I think Doctor Finkeel's experiments will bear out that contention, though I have no opinion on it myself.

BY DOCTOR RHOADS:

Can you give us your figures on that?

BY DOCTOR FRIEDMAN:

Out of three thousand cases, we have only had five

cases of carcinoma and I just sent one case to Doctor Vorwald that we're not even sure is carcinoma.

BY DOCTOR VORWALD:

And I'm sorry to say Doctor Vorwald hasn't examined the sections yet so I can't comment.

BY DOCTOR FRIEDMAN:

It's not likely that we're missing it, because we do extensive diagnostic studies and, in view of your comment, I thought perhaps you might have some other information on the studies.

BY DOCTOR RHOADS:

I think some of this might be clarified further; three thousand cases of what?

BY DOCTOR FRIEDMAN:

Three thousand cases of coal miners pneumoconiosis.

BY DOCTOR RHOADS:

In Wales?

BY DOCTOR FRIEDMAN:

No, in Alabama; I thought you said Mayo Clinic, in Mayo, and we only have either four or five cases.

BY DOCTOR LYNCH:

That's clinically, Doctor?

BY DOCTOR FRIEDMAN:

Pathological diagnosis, and a fifth case is in debate; we're not sure it's a carcinoma or not, because

Doctor Vorwald is one of those to express an opinion.

BY DOCTOR RHOADS:

Then if I may summarize, in three thousand autopsied individuals ...

BY DOCTOR FRIEDMAN:

No, three thousand clinical cases, those in which we have been able to establish a definite diagnosis of carcinoma of the lung, we have only been able to prove it in surgery, and the autopsy table in four or five cases.

UNIDENTIFIED SPEAKER:

Do most of your miners chew or smoke?

BY DOCTOR FRIEDMAN:

They do everything. Well, they smoke an awful lot when they come off the job; they chew; they smoke; they drink, they eat all sorts of food.

UNIDENTIFIED SPEAKER:

Do they smoke as much?

BY DOCTOR FRIEDMAN:

Well, we ask in our social history, we ask the amount of cigarette smoking they do or cigar smoking or tobacco, whatever type of tobacco they use, and I believe it would be safe to say that the average coal miner would smoke between three quarters and one and a quarter packs of cigarettes a day.

BY DOCTOR RHOADS:

Doctor Mider has a comment on these interesting remarks.

BY DOCTOR MIDER:

In what age group did these three thousand individuals fall, did you say?

BY DOCTOR FRIEDMAN:

You're asking me a lot of questions and I haven't prepared statistics on it, but they run - the oldest coal miner whom I took care of, I think, is eighty-six years of age and the youngest one whom I've taken care of as a coal miner is around twenty-four or twenty-five, so you see I have not - I have not taken the statistical analysis, but that's roughly the spread.

BY DOCTOR MIDER:

This brings up the subject that has been brought up before today and that is an adequate frame of reference for judging the incidence of cancer in a population, whether it is excessive, lower than normal or higher than normal. Certainly, we have not had adequate standards of reference in the past. Can you hear me back there?

We have not had adequate standards of reference in the past. In the United States at least, autopsy statistics cover a relatively small proportion of decedents. The use of mortality statistics imply that cancer is not curable. Well, that may be debatable. Cancer now is

reportable by law or by regulation in twenty-eight of these United States and the district of Columbia, and Alaska.

BY DOCTOR RHOADS:

After death?

BY DOCTOR MIDER:

No, cancer. It would seem practical then to derive tables of incidences of anatomical types of malignant neoplasms in relation to both age and sex for the principal types we're interested in and from these calculate the expected occurrence of cancer of these various types in sizeable populations in which we are interested.

Now, the method for that was devised by Curtis and, to the best of my knowledge, it was first published of all places in the American Journal of Obstetrics and Gynecology, in 1946. The New York State Department of Health, through its Bureau of Cancer Control, I think largely due to the efforts of Doctor Levin, has made such tables for the year 1945 to the year 1947. I wonder if he would like to comment on this type of standard of reference which might improve our criteria for saying that this occupational or this environmental group has or has not an increased incidence in cancer.

BY DOCTOR RHOADS:

Doctor Levin, you already showed one such reference in your slide this morning.

BY DOCTOR LEVIN:

Yes, I was going to ask Doctor Friedman if he would mind telling us how long he had been practicing medicine?

BY DOCTOR FRIEDMAN:

I have been practicing medicine since 1941.

BY DOCTOR LEVIN:

That's a period of observation of ten years?

BY DOCTOR FRIEDMAN:

Less than that, because I haven't practiced to make a living since 1945 or '46.

BY DOCTOR LEVIN:

I don't think that's particularly pertinent. In a period of ten years, you wouldn't have observed your three thousand cases for more than a fraction of their life span. Now, if you observed three thousand males, according to the tables Doctor Mider was talking about, according to our data that come from all the hospitals, laboratories and physicians practicing medicine in New York State throughout their life span, in our three thousand cases, if they had neither more nor less lung cancer than were expected, there should be thirty cases.

Now, if you also watched them for ten years, it's quite possible that you observed just a lot of the normal incidence of lung cancer in three thousand men. Of course, if we knew what the age of these people were when you first

saw them, and of course, what the ages were when you last saw them, we could use the tables that Doctor Mider was referring to and give you something a little bit more refined. I think it's a kind of observation that you can not bank on as far as it goes, is worth following up more precisely and may actually lead to something that is a more precise type of data, you can't tell. The tables are available if anyone would like them; you can have them.

BY DOCTOR FLETCHER:

Let me comment once more on this terribly dangerous habit of trying to form statistics on hospital population, and I really do feel - I reproved Doctor Friedman for it yesterday, but he apparently is quite unrepentant.

Doctor Cochrane, - I did describe to you a survey that Doctor Cochrane has done in the Rhondda Valley in South Wales where he has X-rayed a complete mining community. Now, it was very interesting in the course of that X-ray survey where he X-rayed over nine thousand miners and ex-miners, I'm sorry I haven't got the detailed figures here, but they'll be available for the record - he didn't actually pick up on the X-ray a single case of pulmonary neoplasm among that whole population, although he did pick up two intrathoracic - what you - what do you call them, you know, pains?

BY DOCTOR RHOADS:

Mediastinal pains?

BY DOCTOR FLETCHER:

Two terms, you know, it's astonishing. You pick it up, but not a single neoplasm, but within a year of the survey, there had been again -- I'm quoting from memory -- there had been four deaths from that neoplasm in that group. Now, we shall have actual figures for the actual mortality from neoplasm on this actual group of miners extending over the years, with accurate age distribution, and I think we should be able to give a fair answer to the effect of coal dust on the incidence of neoplasm in that group.

In the meanwhile, I think that Professor Gough's experience, with an enormous experience of post mortem work in coal miners in South Wales, he thinks that there is no influence at all of coal dust on neoplasms. He sees just as many neoplasms in miners as in non-miners. But I don't pay any greater respect to that by the evidence from the post mortem department than I do to Doctor Friedman.

BY DOCTOR RHOADS:

Doctor Friedman?

BY DOCTOR FRIEDMAN:

I want Doctor Fletcher to know I did learn yesterday, and that today I didn't make any definite conclusion. I was merely asking a question. I don't think you should have reproved me again today.

BY DOCTOR VORWALD:

During very recent months, years really, I have become increasingly distressed by efforts of some who incriminate a dust which has been inhaled into the lung, deposited in all areas of the lung, which might be seen as present in an area of malignancy as the causation for that malignancy. I refer specifically to the asbestos body which occurs in tumors developing in the lung, and I should like to have someone comment upon the validity of that sort of reference or not. Doctor Mider may like to comment.

BY DOCTOR RHOADS:

Doctor Mider?

BY DOCTOR MIDER:

Doctor Mider doesn't like to comment on that, but it seems to me that it's your possible hope, probably your hope, that sort of argument, and not susceptible to scientific proof or reputation; it's a state of mind, is it not?

BY DOCTOR RHOADS:

Doctor Levin?

BY DOCTOR LEVIN:

Coming back to Doctor Mider's very interesting paper, I wonder if you would mind commenting on this question which may be unfair. In view of the facts regarding experimental pulmonary neoplasms in animals such as the mouse, what justification could you educe for drawing any

conclusions whatsoever regarding the effect, pro or con, of various agents in the mouse, in what way would that have any bearing on the neoplasm in the man, in view of the marked differences that you have?

BY DOCTOR RHOADS:

There is a question for you.

BY DOCTOR MIDER:

It seems to me about the only information that we can obtain from the mouse and man, in relation to man, is that information which has afforded us basic factors of information on carcinogenesis and is duplicated in some measure by what little we know about carcinogenesis in the human species and in other species.

In general, with some very definite exceptions, comparable approaches in man and in the mouse have led to comparable results. We must remember that the experimental cancer approach came out of the clinic and that, as far as we know today, there seem to be only five unadulterated substances which are capable of causing cancer in man that we know of, X-rays, ultra-violet rays, trivinyl arsenic, benznaphthalene and more recently, benzadrene.

Each of these agents is alleged - and I think with adequate evidence - to produce one or more types of neoplasms in experimental animals. Though one can not differentiate from one species to another directly without some

evidence that this is one, I do think that the study of pulmonary tumors in the mouse and of other tumors in other species have contributed to our basic faculty - our basic factual information as to what to expect in searching for human carcinomas.

I wish Doctor Smith would interject some remarks here. He knows a great deal more about this situation than do I.

BY DOCTOR SMITH:

I think the difference is that I don't know anything about it. I'd like to say that the reference Doctor Rhoads asked for and very properly called to your attention, that is the abstract by Lithgow and Finkel, describing tumors in the lungs of animals that have been allowed to inhale radioactive serum, is an abstract that I have - that I read carefully and couldn't understand. I believe if you look it up, you may encounter the same difficulty, so I wrote to Doctor Lithgow and he replied that - I asked Doctor Lithgow if he meant that he had, in fact, gotten tumors and if so, what sort of tumors he got. He replied that he did mean that he had gotten tumors and that they were epidermoid carcinomas.

Now, since he was talking about a matter of pathology and not a statistical question, I was inclined to accept what he said as fact, and I think it stands as an

extraordinarily important advance, because it represents, inasfar as I know, the first experimental success in producing in animals the type of carcinoma that forms a really major segment of the lung cancer problem in man, and as far as commenting on that general problem goes, I think that we are right there faced with a very important comment on it, in that here we are talking about an experiment which is so recent that it is in literature only in abstract form and yet the statisticians tell us and the clinicians have long known that lung cancer has become perhaps the outstanding clinical problem in males. We are faced with a real and a serious situation, and as far as our ability to study that situation experimentally goes, we have very little experience to guide us and that we urgently need more studies of suspected carcinogens by isolation techniques.

BY DOCTOR RHOADS:

Well, that's an arbitrary statement, Doctor. I would like to, with all deference to Doctor Mider, you referred to the five carcinogens which have included man, all having shown activity in the experimental animals. Did you yourself not question the animal experiments with arsenic?

BY DOCTOR MIDER:

Oh, excuse me, that's right.

BY DOCTOR RHOADS:

Are there other comments? (No response). If not,

then we will proceed with the next paper. Doctor Anna Baetjer, Pulmonary Cancer in Experimental Exposures to Chromium.

BY DOCTOR BAETJER:

Oh, don't you want to give them a little rest first? After all, they don't all have rubber rings like I do.

BY DOCTOR RHOADS:

She says they don't all have rubber rings like she does, so don't you want to give them the rest period. We'll will take a short recess then.

(Recess from 4:20 to 4:25 P. M.).

BY DOCTOR RHOADS:

Will you take your seats, please, so we can go on with the program. Will you please be seated; will you please be seated so that Doctor Baetjer can be heard? Gentlemen, will you please be seated? Our next speaker would like to be heard and we'd like to hear her.

Will the meeting please come to order. The next paper is by Doctor Anna Baetjer, Associate Professor, Environmental Medicine, Johns-Hopkins School. She will discuss pulmonary cancer in relation to exposure to chromate. Doctor Baetjer.

BY DOCTOR BAETJER:

Doctor Rhoads, Ladies and Gentlemen: First of all, I'm going to have to justify why I should stoop to do

Chapter Twenty

Pulmonary Cancer in Experimental Exposures to ^{Chromate} Gaseous Chromium

Anna Baetjer, Sc. D.

First of all I'm going to.....

.....

.....on this subject.

Pulled

B. 1.

following Anna Baeyer's
Paper.

"Exp. Studies. on Pul. Also. In
Relation To Cholesterol"

BY DOCTOR BAETJER:

Doctor Rhoads, Ladies and Gentlemen: First of all, I'm going to have to justify why I should stoop to do

THE JOHNS HOPKINS UNIVERSITY
DEPARTMENT OF ENVIRONMENTAL MEDICINE

SCHOOL OF HYGIENE AND PUBLIC HEALTH
615 NORTH WOLFE STREET
BALTIMORE 5, MARYLAND

PLEASE REPLY TO UNDERSIGNED

November 24, 1953

Dr. Arthur J. Vorwald
163 Park Avenue
Box 509
Saranac Lake, New York

Dear Art:

I have another change to make in the chromate paper. The rat experiments which we are repeating for the study of lymphosarcomas, appear, at this time, to be diametrically opposite from the first experiment. We have had a large number of controls dying of lymphosarcoma and no experimentals. In view of this, I wish to change the bottom of page 5 and number 3 of the conclusions on page 6. In order to make this as simple as possible, I am sending you the corrected pages 5 and 6. I have added a note also about the zinc chromate experiments. Since Dr. Steffee reported these at the cancer conference in New York, I have decided to put this result in my paper.

I will certainly appreciate if you will change these two pages in my manuscript.

Sincerely yours,



Anna M. Baetjer, Sc.D.
Associate Professor

AMB:bvb
Enclosure

Each experimental group was compared with its control group and the observed differences were tested for statistical significance.

b. Results

The experiments are still in progress and the statistical analyses of the data have not been completed. Hence, the results reported here are only tentative.

In the individual mouse experiments, the percentage of mice with tumors and the average number of tumors per mouse were not significantly higher in the experimental groups than in their respective control groups. However, when the various experiments were standardized for age and combined, the incidence of tumors was significantly greater in the Swiss strain mice which received intratracheal injections of the mixed chromate material suspended in olive oil, than in the control mice which received intratracheal injections of saline or which had no treatment. This difference was not apparent in the A strain mice, but a significantly higher incidence of tumors resulted from the intratracheal injection of olive oil alone in this strain. It is possible that the higher incidence of tumors in the Swiss strain was in part due to the olive oil and in part to the chromate materials. The other characteristic finding which resulted from these experiments was an epithelial investment of the alveolar walls in all of the strain A mice and part of the Swiss mice which had been injected intratracheally with zinc chromate. No bronchogenic carcinomas resulted from exposure to the chromate materials in any of the mouse or rat experiments.

A number of other pathological changes, some of which were attributable to the chromate materials, were observed in the mice and rats in these experiments. These changes are now being studied by the pathologist.

c. Conclusion

The pathological studies and the statistical analyses of these experiments have not been completed. The tantative results from these experiments at this date are:-

1. The exposure of mice and rats to chromate dust in a dust chamber and the intratracheal injection of chromate materials did not cause bronchogenic carcinomas in these species of animals under the conditions of these experiments.
2. The intratracheal injection of olive oil and of the mixed chromate dust suspended in olive oil appeared to cause an increase in the incidence of pulmonary adenomas in certain strains of mice.
3. The intratracheal injection of the complex zinc chromate caused a metaplasia of the epithelial cells lining the alveoli.

THE JOHNS HOPKINS UNIVERSITY
DEPARTMENT OF ENVIRONMENTAL MEDICINE

SCHOOL OF HYGIENE AND PUBLIC HEALTH
615 NORTH WOLFE STREET
BALTIMORE 5, MARYLAND

PLEASE REPLY TO UNDERSIGNED

August 17, 1953

Dr. Arthur J. Vorwald
P. O. Box 509
Saranac Lake, New York

Dear Art:

Enclosed is my paper and the corrected discussion.
I am sorry it is in such poor shape, but I did not have
time to do anything better.

Sincerely yours,

Anna M. Baetjer per BVB

Anna M. Baetjer, Sc.D.
Associate Professor

AMB:bvb
Enclosures 2

August 28, 1953

Dr. Anna M. Baetjer,
Department of Environmental Medicine,
School of Hygiene and Public Health,
The Johns Hopkins University,
Baltimore, Maryland

Dear Anna,

By this time you are, no doubt
climbing around the Andes of South America
and enjoying Lima, Santiago and Rio. I hope
that you find it most interesting and that the
altitudes will have little effect upon your
respiratory abilities.

We have received your corrected paper
and discussion for the Symposium. All of it is
in perfect shape and I am grateful for the time
and effort which you gave to it.

Sincerely yours,

Arthur J. Vorwald, M.D.

usually a very short period of time. ^{Some} Many of our cases, on coming to ^{the} our hospital dispensary, claimed that their severe symptoms didn't begin until two weeks before ^{they} ~~these~~ individuals came to the clinic, and they could ^{we} ~~not~~ be diagnosed as having lung cancer. So far as we have been able to determine, the clinical symptoms and the pathology are no different for lung cancer in chromate workers than for lung cancer of other origin.

DR. RHOADS: What brought these patients to the clinic? Symptoms referable to the lungs?

DR. BAETJER: Some came with lung symptoms ^{such as} or pain in the chest. Some patients ^{to} about whom I have read came with symptoms referable to metastases.

DR. RICHARDS: Is there any hazard after the material has been shipped out?

DR. LANZA: So far as I know, there has been no complaint.

DR. HUEPER: In regard to the possibility that the material shipped out of the plant will become carcinogenic, there have been reports from Germany that zinc pigment - zinc chromate - workers have developed carcinoma. We are attempting to find out whether the painters who sprayed zinc chromate on airplanes during the last war have an increased cancer incidence.

See Substantiated attached

DR. BAETJER: There have been reported in the literature 134 cases of lung cancer associated with an exposure to chromium. Of those cases 121 were in the chromate producing industry, 10 in the chrome pigment industry - the manufacture of zinc chromate and lead chromate pigments - and only 2 ^{cases} among people who use chromium. There was one case in Baltimore in a chrome plater who was exposed also to many other substances and there was one who sprayed chromium (or sprayed with chromium) paint. Thus, of the 134 cases there were 132 in the chrome producing or chrome pigment producing industry and only 2 among chrome users.

heard of any cases of

We have never had any cancer of the skin in chromate workers nor cancer of the nasal septum, even though a large percentage of the workers have perforation of the nasal septum, and ulcers of the skin due to the chromium chemical.

It is interesting to review the duration of exposure of these people to chromium before they ^{developed} get cancer. The average age of these individuals was 52 ^{at the time of death} years and the range of ages was from 31 to 72 years. The duration of exposure varied from 4 to 47 years ^{with an average of 18 years} and the interval between the initial exposure and the diagnosis of cancer was about 17 years, with a range of 4 to 47 years. In some cases cancer may develop many years after the termination of exposure but the interval between diagnosis and death is, as in all cases of lung cancer,

Pulmonary Cancer in Experimental Exposures to Chromium

First of all I'm going to.....

on this subject.

DR. RHOADS: What method was used to measure the chromium?

DR. BARTJER: It has been extremely difficult to analyze tissue for chromium because most available methods are not good. [We used a German method, modified with the help of Dr. Hueper, to whom we are grateful.]

The material is wet ashed and the hexavalent chromium is reduced to the trivalent form. The material is then ashed in a muffle furnace. Iron, which is a complicating factor because it carries down chromium, is kept in the trivalent form and precipitated with bromine; at the same time chromium is brought back to the hexavalent form. The color is then determined with bromine.

DR. RHOADS: Was the concentration of dust used for the animal experiments about the same as that existing in the plants, or was it higher?

DR. BAETJER: Nobody knows what the concentration was in the plants at the time these men ^{where} ~~got~~ ^{were} ~~their~~ ^{ad} exposed. The concentrations that have been determined by the Public Health Service are somewhat comparable to those of our experiments; for the ~~nice~~ experiments we employed concentrations ten times,

and for the rat experiments twenty times, the maximum allowable concentration. These conditions represented about the highest level of tolerance for the animals; in fact, we killed many of them by trying to ^{determine} (expose them at) the maximum level. The dust concentrations used were higher than those in the plants at present but were much lower than the concentrations in the plants years ago. In the old days the dust concentration, as reported in the literature, ranged from less than 0.1 mg. per cubic meter of air to 50 milligrams. We were working in the range of about 1 to 3 milligrams.

DR. RHOADS: You were working with animals exposed to a concentration which was sufficiently high to make many of them fairly sick?

DR. BAETJER: Yes, but we reduced the concentration to the point at which they would survive. Growth was impaired, in some groups quite definitely, and in others only slightly. The weight was down, but not very much.

DR. MIDER: Were there pathological changes other than neoplasia in these animals?

DR. BAETJER: Yes. The inhalation mice showed very marked differences. The inhalation rats exhibited a high degree of inflammatory reaction. In the mice injected intratracheally with the mixed dust, so that a definite measured dose was put in each animal, there was marked fibrosis with atelectasis. Since these changes were at the site of injection, we knew the injected material had reached the lungs, but the weight gain of those animals and of the controls did not differ substantially.

The barium chromate mice showed no difference at all, except possibly a slight irritation.

DR. KUSCHNER: In your experiments did you produce lesions comparable to the perforation of the nasal septum that occurs in industrial workers?

DR. BAETJER: There was nothing of that nature in the rats or mice but we are now getting perforation of the nasal septum of rabbits. We had no evidence of any malignancy there. *of the nasal septum*

DR. HERMAN: In a recent case report it was alleged that liver damage in a worker using chromate - I believe he was employed in a plating industry - was caused by his industrial exposure. It has been alleged that lesions have been produced in the kidney also. Would you comment on liver or kidney lesions?

See substitute which reduced these paragraphs to one sentence

~~DR. BAETJER: I would not like to comment because we have not studied those other features. I have not found in the literature any statement that chromium causes liver damage. There are many reports in the literature of kidney damage produced experimentally in animals by the injection of chromates. Dr. Riegler conducted some of those studies and Dr. Hueper made some also.~~

DR. HUEPER: There was in the J.A.M.A. a paper about liver damage after exposure to chromate.

~~DR. BAETJER: The J.A.M.A. paper, in my opinion, presented an extremely weak case for chromate. Liver damage ^{was reported in} to one worker in the chromium industry was reported and, therefore, the damage was attributed to chromium. Four persons working nearby exhibited slight changes in the liver test ^{function} but they had no other symptoms. *I do not think that this paper presents any real changes whatsoever. It seemed to me that the paper was a very poor one. Proof that chromium compounds cause liver damage*~~

DR. KUSCHNER: Dr. Baetjer stated that the pathology in these cases of chromate carcinoma was identical with that found in the garden variety of carcinoma. To pathologists and clinicians lung carcinoma is, in many cases, unicentral and with Lindberg's study on the genesis of carcinoma there have been many changes

in many portions of the bronchial epithelium. Some persons assume that this unicentral character is an argument against the importance of inhaled carcinogens. I do not believe that is necessarily so, but I would like to know whether in these chromate carcinomas there are multicentric tumors or metaplasia or perhaps carcinoma in other sites or demonstrable effects on the whole bronchial tree.

DR. BAETJER: The carcinomas reported in the case histories that I have read were localized. Sixty-eight per cent ^{of our cases} were in the right bronchial tree; in the German series 60 per cent, I believe, were in the right bronchial tree. In all these cases the carcinoma was localized and was not distributed throughout the bronchial tree.

DR. KUSCHNER: Were there extensive studies of the bronchial tree?

DR. BAETJER: No, so far as I know. ~~I do not believe that anything has been published about the results.~~

DR. MEREWETHER: Dr. Baetjer, did you have any cases of isolated carcinoma of the lower lobes? Professor Cough told me, about seven years ago, that he had had one or more cases that had nothing to do with the bronchi or bronchial nodes.

DR. BAETJER: ^{of} All my cases were ^{bronchogenic w origin} ~~definitely concerned with the bronchi~~, with the exception that in one case there was, in addition to the bronchogenic cancer, a pleural cancer of different type in the lower lobe, but, according to the pathology report, they were all related to the larger bronchi.

animal experiments and why it's worthwhile at least why I thought it might be worthwhile. Actually, I thought I'd already done a good enough statistical and epidemiological study so that I had proved, along with Doctor Machle and now the Public Health Service that chromates do produce or at least are a factor in the production of lung cancer in man. At least, my epidemiological study passed our statistics and epidemiological department and I defy even Doctor Levin to say that didn't contain all the quirks that a statistician can suggest first, so now I'm going to tell you about these animal experiments.

We felt that if we could possibly reproduce this condition in animals, then we would have a basis to start to study the changes in the tissues that result when a simple substance, if you could prove it, causes cancer. We also hoped that we could tell exactly which substance was the carcinogenic agent in these human exposures if we could reproduce it in animals. That then - so that we thought it was well worthwhile setting up a large project to study the effects of chromium compounds on animals.

This work was supported very generously by the National Cancer Institute and has been a project in which Doctor Louny of the United States Public Health Service, Department of Pathology, and Mr. Udas on our staff, have participated with me.

I would like to divide my presentation into three parts: First, animal experiments on our efforts to produce lung cancer in animals; secondly, our results of our animal - our analysis of two cases of humans who have been exposed to chromates, and a comparison of that with the results that have been reported in the literature; thirdly, as time permits, I might hazard a guess or two as to why or how this chromium might act, and which substance -- no, I'm not going to guess that, because I can't -- I might comment on what chromium might possibly do to tissues, and I might speculate as to which substance might possibly be responsible.

Taking first then our animal experiments, in the literature, there are only a few studies which report the effects of chromium compounds or attempts to produce cancer with chromium compounds. Lukanin exposed seventeen animals in a plant for eight months. He got some quite marked pathological changes in the lungs of the animals, but he did not mention cancer, and the description of the pathology does not resemble cancer.

Doctor Snipkin, injected the iron chromite or the chromite ore into cancer susceptible mice, and he found that there was no increase in the spontaneous lung tumors of these mice. Furthermore, he found that the injection of chromite dust intravenously into animals did not increase the effect of metachromatin in the production of lung tumors.

The third paper which I think is worth mentioning is a paper by Shintz in which he implanted into the trochanter of animals, a few animals, metallic chromium. He reported that after four years, one animal developed a lung cancer which was a carcin... a sarcoma, that one and possibly a second bone cancer occurred at the site of the implantation.

The other work that has been reported in the literature has been not worth anything, because it has involved only one or two or three animals. That was what was in the literature.

Today you heard Doctor Hueper say that he had been able to produce three lung cancers in seven rats with the chromite ore. Such was the background in which we started.

Our experiments have been of much greater magnitude than any of these which I have reported. First of all, with regard to the character of the animals which we used. We used mice and rats, three strains of mice, a cancer susceptible inbred strain, the A strain that you have just been hearing about; secondly, a Swiss strain which has a moderately high incidence of spontaneous lung tumors, and thirdly, the CO-57 inbred strain which has very, very low incidence of spontaneous lung tumors. The other type and species of animals which we have used so far are rats and in that, we used a mixed breed, a single strain that has

been in our laboratory for many years. So much for the type of animals.

What about the type of exposure? We used, in the first place, inhalation exposure over periods of four hours per day for five days per week for shorter and longer periods of exposure up to the life term of mice and rats in some cases. These animals were put in a dust chamber and thus exposed by inhalation. The second method of exposure that we used was intra-tracheal inoculations with the material. These were given repeatedly in mice and in rats. The third type of material which we used, the third method of exposure that we used was by intrapleural injection. The fourth type of exposure that we used was intravenous injection of material.

Next, with regard to the type of material which we used. We wished to reproduce in our dust chamber the conditions exactly as closely, similar, to that condition which exists in the chromate chemical manufacturing plants where these cases of lung cancer have occurred. We asked one of the plants which has been most cooperative and has supplied us with material and the analysis of our materials, in every instance in which we have come to them.

The material which we used in the dust chamber was a mixed dust. I have put on the board here, if you can't hear in the back, please say so -- I have put on the

board here, the material which we have used. In the industry, in the chromate chemical manufacturing industry, the chromite ore, that 's the chrome iron ore, is heated with soda lime ash to form a rose material. This rose material contains about six percent chromium, in the form of sodium chromate. It contains an intermediate product, the sodium chromate is the end product. It's water soluble. The intermediate products are water insoluble but soluble in acid and it also contains a small amount of unchanged chromite, which is insoluble or relatively insoluble.

This material - the chromite - has the chromium in the form of a hexavalent, in a trivalent form. The sodium monochromate is of a hexavalent form. The intermediate products are partly trivalent, partly hexavalent and the unchanged ore, of course, remains as trivalent.

We took, for our inhalation experiments and for some of our intra-tracheal experiments, this mixed material, because we believed that this represented, largely, the exposure which existed in the plants, and so we had this material which the plant ground for us to a small particle size. However, the large part of the chromate in this material of the hexavalent chromate is almost entirely in a water soluble form. It, therefore, had to be in the intra-tracheal injections and intra-pleural injections, put in oil. We therefore used for our control animals, the

oil without the chromates, the chromate mixture in oil for the experimental animals. This, therefore, was not suitable for intravenous injection.

Furthermore, we have already heard that there have been some cases in the chrome pigment industry, in which there were primarily zinc and berium and red chromate. We, therefore, used as our second type of material, zinc chromate. It's this very complex form that you see here on the board, is the type which is the zinc chromate pigment. For control experiments, we used zinc carbonate. We also used - and this is relatively insoluble, but does break down slowly, yielding a potassium dichromate and a zinc complex.

We also used berium chromate and for the controls for that, we used berium sulphate. We used the zinc and berium chromates for the intra-tracheal and intravenous injections.

I'll turn this off temporarily; we'll come back to that again. Therefore, we used these three materials, the mixed dust, the zinc chromate, and the berium chromate. All this dust was in a particle size such as that - such that at least eighty-five percent was one micron or smaller in size, so that we're dealing with practically comparable dust as far as particulate matter is concerned. So much for the material which we used.

Now, with regard to the animals and their exposure. We used a concentration which was just below the lethal point. We tried to get as high a concentration as possible. Some of the animals, however, died, because of the toxicity of the material or for other, incurring infections or other reasons, and the results which I want to report on the mice are limited to those which survived to the point where we killed them.

The rat experiments which were only inhalation and intra-tracheal with the mixed dust, we counted all animals which survived one year and we killed the final lot which survived to a two-year period. We concluded that the - in the rats, those which died then after one year of age. So much for the materials and the type of exposure.

We - in the mice experiments which you have heard from Doctor Mider, we did spontaneous experiments, we used - when the animals were killed, we examined the lungs for the incidence of spontaneous tumors or tumors that were visible on the surface. We autopsied all animals, the dead ones too, but those I can't report to you because we have not yet studied them pathologically.

The animals were killed at various age periods. This was necessary in the mice because of the development of spontaneous tumors, and we wanted to determine whether the tumors appeared earlier in our chromate animals than they

did in the controls. We killed the animals, therefore, at various intervals. We took out the lungs, counted the number of tumors on the surface of the lungs and also made cysts, histological sections, I should say, of these tumors for confirmation of their pathology. All other abnormal conditions of the lungs were also sectioned and studied by the pathologists. All other organs were examined and where there was any evidence of other types of malignancies in other tissues, they were also sectioned for histological examination.

The animals were then compared with regard, in the case of the mice, with regard to the number of animals which survived, which had pulmonary tumors on the surface, the number of or the percentage, that is the percentage of animals with tumors, the average number of tumors per mouse, the early or late onset of tumors, the presence of multiple tumors; as Doctor Mider has said, you get often many tumors with the carcinogens. We determined, therefore, the percentage with multiple tumors, compared the controls and experiments also with regard to other types of lung pathology and other types of malignancies.

One thing I forgot to say which is important, and that is that we had to maintain our weight gain of our experimental mice to be equivalent of the control mice, to be equivalent to the experimental mice, because it has been

shown that caloric intake may effect the incidence of lung tumors. All of our mice, therefore, were weighed and the controls' diet was restricted so that it was - the weight gains were equal.

After we killed our animals, made the sections and studied them by pathology, we then compared the experimentals and the controls in these various methods that I have said. In addition, we analyzed the lungs for the presence of chromium to be sure that we were getting chromium into the lung tissue. So much for our methods.

Now, about the results of our animal experiments. In the mice experiments, altogether, we used large numbers of mice such that at certain - at the date of killing, we had a total in all of our twenty-five experiments of about sixteen hundred mice. This yielded us data which we could treat statistically. The - in regard to the incidence of lung tumors, their age at onset, the average number of tumors and the character of the tumors, there was essentially no difference in the control groups as compared with their - in the experimental groups as compared with their respective controls.

The results, therefore, of the mice experiments with regard to the incidence of pulmonary tumors, was on the whole, negative. There also was no difference between the incidence of other malignancies in the mice, between the

experimentals and the controls.

In regard to the other pathology, however, we found some characteristic changes with one of our materials. With regard to the mixed dust, the mixed dust caused a slight inflammatory reaction. In the intrapleural animals, it caused a marked fibrosis, a marked inflammatory reaction, but in inhalation and the intra-tracheal experiments, there was only a slightly greater evidence of some irritation in the lungs in the experimentals versus the controls.

The berium chromate also produced only a slight irritation of the lungs, both in the intra-tracheal and intravenous experiments. However, the zinc chromate proved much more interesting. In the intra-tracheal zinc experiments, the mice ' lungs showed a metaplasia of the alveolar epithelial lung. I can not report further on this, because that study has not yet been made by the pathologist.

Also, in the zinc chromate, we injected intravenously, produced a marked inflammatory reaction in the lung tissue in a high percentage of the animals. Thus, the zinc chromate proved, by far, the most irritating and by far the most damaging of the materials by intra-tracheal and intravenous routes. The mixed dust was very irritating in the intrapleural rote. So much for the mice experiments.

Now, about the rat experiments. We started the inhalation group of rats with a large number. Unfortunately,

we had a large number of deaths, both in the intra-tracheal and in the inhalation group of rats. The intra-tracheal animals died so rapidly we were forced to kill them at an age of forty-eight weeks, and except for the irritative changes, there was nothing significant. The group in the inhalation chamber, which was exposed to the mixed dust, however, did show one possibly interesting feature.

These animals - there were fifty-eight that survived a period of sixty-eight to one hundred and one weeks in the dust chamber. These rats, when they were examined, showed, out of the total fifty-eight, three definite cases of lymphosarcoma and two possible cases of lymphosarcoma. Of those lymphosarcoma cases, one appeared to be primarily in the lung, one possibly primarily in the lung and one was secondary.

We had fifty control animals left, but fortunately, we had in our laboratory a large number of stock animals of the same age, same strain, same diet, as our control animals. Therefore, we were able to increase our control group to three hundred and fifty-six animals, living on the or to the same age as the experimental group, and proportionately killed at the same age period. Of the three hundred and fifty-six, we had five animals which showed malignancies, none of them were in the lung and none of them involved the lung tissue.

If you take the percentage, therefore, of experimental animals, which had malignancies, as in the experimental as compared with the controls, and calculate the differences, divided by the standard area of differences, using Yates Corrections, because we are dealing with numbers under ten, you find that the X over sigma value, using three experimentals and not including the two questionables, three experimentals and five control malignancies, you come out to an X over sigma value of 2.4. If you take the malignancies which effect the lung tissue in the controls, three animals as compared with zero in the experimentals, you come out to an X over sigma value of 3.5 or so.

This shows that they are, according to statistical figures, statistically significant. But I'm not going to say anything on three animals, no matter what the figures show. I don't like drawing any conclusions on just three animals. That doesn't go right, so, as a result, we are repeating the experiment if we can repeat it, and we hope that we will be able to keep the rats in larger numbers in the dust chamber this time, so that we do not have as many deaths and we will have more at the end, we hope we might be able to confirm it. At the present time, I would not say we had proven anything at all, but we have a suggestive lead, as indicated, that we might repeat the experiment.

The rats, therefore, showed that type of reaction.

The mice showed essentially negative. So much for our animal experiments.

The second part of our work has just begun and that is that we are now analyzing tissue for chromium. Whenever we can persuade anybody to let us know that they have one of these men who had worked in the chromate chemical producing industry, with - which is coming up either for autopsy or for pneumonectomy, we immediately say, let us have some of the tissue. To date, we have gotten two.

One was a body that was disinterred, and of a man who had had a year and a half of exposure, which was terminated two years before his death, who did not die of lung cancer. The other is a case that came into our hospital, a couple of weeks ago for - with symptoms of chest pain. They diagnosed it as pulmonary carcinoma, operated within two weeks, the man had, therefore, his exposure up to sometime within a month of the time we got the tissue for analysis.

I'd like, therefore, to show you the results of our two cases and to compare those with the results of the other analyses which are only four, that have been reported in the literature.

First of all, I might, in the lungs analysis work, I might tell you what normal results have been reported. Chromium appears to be present in almost all or may be present

in almost all types of normal tissue in traced amounts. B. Warren Benz, many years ago, showed that tumors just taken from the autopsy room or from the operating room, many of them showed traces of chromium, but did differentiate between benign and malignant, present in some, benign, in some malignant, some benign; present in others, of both types. They could not decide on any possible function of chromium in these cases.

Chromium also appeared in other tissues of normal people. It appears sometimes in the urine, sometimes in the blood. As far as I have been able to make out, we had no idea that it has any physiological function. Thus, I put in the first table, a few results. All right, if we could have the table.

These are the analyses that have been reported. I might say the other people who have reported these cases, which is Mancuso and Hueper and United States Public Health Service, this represents four cases and our lung case, ours put in terms of fresh tissue -- our disinterred body we couldn't compare, because, of course, we could only do it in ash, so that this represents our case of the man with thirty years exposure who just came to operation for pneumonectomy.

Now, there are a couple of things that interesting and that's why I'm taking your time to show you these

figures. In the first place, the chromium, the chromium in the lung tissue is relatively high as compared with what has been reported to occur in one or two normal cases. Of perhaps greater interest, because you expect this in the chromate workers, and by chromate workers, I mean always people in the chromium chemical manufacturing industry, not people using chromiums, it is relatively high. You would expect that, but notice we found a high incidence of chromium in your lung tumor, but there are two cases on record in which there was no chromium in the lung tumor in chromate workers, although other people have also found, in other cases, a high incidence.

You will notice that blood, in our chromate workers, contains chromium, but chiefly located in the red blood vessels. I'll come back to that in a moment. Much less in the plasma. The other point that is of interest in this table is that there are cases of chromate workers, and this represents a large number, a large series of Mancuso's and Hueper's work, in which there were a number, in which there was absolutely no chromium detectable in the urine. I must say we couldn't believe that when we saw it in the literature, but when we got this case the other day, he had no chromium detectable by our methods in the urine either. So much for the distribution in test tissues.

The next slide shows the distribution by lobe of

the lung, in our two cases, expressed as micrograms of chromium per ten grams of ash. In the right upper lobe, where in this - this was the case that came for pneumonectomy, this is the cancer case, this is the lung cancer case. We found a tremendously high value of chromium in the lung where the cancer was located. Here is the cancer which shows twenty thousand micrograms, the immediate surrounding area was fifty-nine thousand; the other lobes were shown here. This man had a right pneumonectomy.

A non-cancerous case is shown here. You will see that in both of these cases, there is more chromium in the upper lobe. Hueper and Mancuso also found that in one of their papers, but one of the German papers found more in the lower lobe, I don't know whether that was true. We found a higher chromium deposit where the cancer was present; one of the German papers claims that was not true in one of their cases.

However, this gives you some idea of the marked area of distribution. In line with Doctor Pratt's comments yesterday that you ought to do the whole lung, I submit or I might say that these tissues were done per gram of wet weight, per gram of dry weight, per gram of ash, so that we have taken into account, on our distribution, all the possible factors of infection and other subsequent pathological changes which might effect the weight of our tissue.

Therefore, we have certain very definite distribution, very marked variations in the distribution of our chromium tissue. Our animal tissues all showed, lung tissue also showed, chromium of about one tenth as much as we got in the humans. So much for that part.

I think I have one minute more, perhaps, is that right, in which I might speculate just as well, so that I won't put my foot in it. There are two things I'd like to talk about. One is, what is the material which might be responsible.

Doctor Hueper indicated to you this morning that he thought it might be chromite dust, and you will see from here that it might be this material which is quite insoluble. I would like to call your attention to the fact that the United States Public Health Service made a study of a plant which uses chromite, that is the chrome iron ore, and in which the concentration was found in the atmosphere of the plant. The mortality records of that plant showed no higher incidence of lung cancer than in the population as a whole, so that we have, on the one hand, Doctor Hueper's experiments, on the other, the United States Public Health Service field study.

The United States Public Health Service has suggested that this material which is the water-insoluble, acid-soluble material, may be responsible for the lung

cancers. They, - this is only a suggestion, they have no real proof. They say that in - in England, in this country, in most of our plants here, this intermediate product is re-worked. In the English plants, it is not re-worked. There has not been a high incidence of lung cancer reported in the English plants. Therefore, they think it might be because this is not re-worked and thus the exposure to this material is less.

I should like to comment on that and say that I have been told by the chromate industry that there is a plant in Germany which does not re-work this material, which has exactly comparable features to the English process, and they have had lung cancer. It's one of the German plants where some of these cases came from. So it may or may not be that material. We have a small amount of it always in the plant in the air.

The majority of workers, including Doctor Machle, have thought that the hexavalent chromates are the responsible factor. They are insoluble, it is true. They are water - I mean they are water-soluble, it is true, and - but, however, they are very corrosive, they are highly oxidizing agents, because of their hexavalent form, and thus they are materials which you would expect to cause a marked reaction in the lungs.

Therefore, we can not say what the material is,

because we don't know. You can take your choice. Your guess is almost as good as mine.

The last point that I would like to make is, what happens to chromium when it's taken into the lung? I don't know at all. I have no idea, but I would like to briefly tell you some very interesting experiments that have been done at Harvard by Bray and Sterling. They showed that if you inject radioactive antibiotic hexavalent chromium intravenously into animals, the material passes immediately into the red blood cells and combines with the globin protein of the hemoglobin. The trivalent chromium on the other hand, does not penetrate the red blood cell membrane/ It is, however, if the red cells are hemolyzed and the hemoglobin is liberated, it will then combine with the globin fraction. If you inject the trivalent form, it will however, combine with the blood proteins.

On the basis of this, it has been suggested that possibly the failure to find chromium in the urine, in some cases, may be due to the fact that the chromium, although it's present in the blood, may be bound to a protein molecule and thus too large to pass through the glomular capsule. Nobody knows what happens to chromium.

Even the tank industry doesn't know exactly the reaction between chromium and proteins. It is known that chromium becomes combined with the proteins, but even under

or in the tank industry, people don't know whether it combines with the carboxyl or the amino group.

So that we who are dealing with tissue at an entirely post mortem, which exists in the body, and who are dealing with live material and not other material, are even harder pressed to determine what happens to the chromium. Very little work has been done on the effects of chromium on enzyme systems. I do not think it yields enough of interest to this group to review it.

In summary then, I would say that we have attempted animal experiments which have yielded us almost no results. In line with - as Doctor Mider said, or in line with most of the other experiments, we have not been able to reproduce in these two species of animals, the type of cancer that we find in humans. We are hoping, by our analyses, eventually, to have something of the distribution of chromium and what takesplace. The results, therefore, have not yielded us anything to really contribute to the sum of our knowledge on this subject.

(Applause).

BY DOCTOR RHOADS:

We can certainly all congratulate Doctor Baetjer and the Public Health Service on a most comprehensive and conclusive study. I would like to ask one or two questions for my own interest. I presume - could you tell us what

method was used in measuring the chromium?

BY DOCTOR BAETJER: Yeah, we used a method - well, that's one of the great reasons why it's been extremely difficult to analyze tissue for chromium, is because most of the methods are not good. We have taken the method suggested by Germany, and have made a number of modifications for which we are very grateful to Doctor Hueper for their help in this.

The material is wet ashed, is not processed, the hexavalent chromium is reduced to the trivalent chromium. It is then further ashed in a muffle oven. It - in order to get rid of the iron which is the complicating factor and which is the very difficult factor, because you get your chromium being carried down with the iron, we try to keep it in a trivalent, we keep it in a trivalent form and precipitate the iron out with bromine.

Then we precipitate out the iron and, at the same time then, bring the material back to a hexavalent form. Then, following that, having it back in the hexavalent form, we determine color with the bromine.

BY DOCTOR RHOADS:

Do I assume that the concentration of dust employed with the animals was about that achieved in the plants or higher?

BY DOCTOR BAETJER:

Well, nobody knows what the concentration was in the plants at the time these men got their exposure. The concentrations that have been measured by the Public Health Service are in the range that occur when they made their summary, and which we have been able to lean on for the few measurements that have been made in recent years, and they are somewhat comparable to ours.

Our concentrations for the mice experiments were ten times the maximum allowable concentration; for the rat experiments, they were twenty times the maximum allowable concentration. They helped to represent the maximum for the animals, in fact we killed a lot of them by trying to get them up just to the maximum levels. It is, therefore, comparable and higher than the plants now have. It is lower than - by far, than the plants in the old days. In the old days, it went less than one tenth of a milligram per cubic meter of air up to fifty milligrams reported in the literature. We were working in the ranges of about one to two to three.

BY DOCTOR RHOADS:

There is a question here that bothers me a little bit, and bothers Doctor Mider a little, I suppose. You were working with animals that had high enough concentration to be fairly sick in many instances, I take it?

BY DOCTOR BAETJER:

Yeah, only we cut it down to the point where they did survive.

BY DOCTOR RHOADS:

You cut it down to the point where it wouldn't impair the health?

BY DOCTOR BAETJER:

We cut it down to where they would survive.

BY DOCTOR RHOADS:

Did it impair their growth?

BY DOCTOR BAETJER:

In the inhalation chambers, they were absolutely lower, in some groups they were only lower, in some groups they were slightly lower.

BY DOCTOR RHOADS:

Was the caloric intake cut way down?

BY DOCTOR BAETJER:

The weight wasn't down too much.

BY DOCTOR RHOADS:

You said you cut it down?

BY DOCTOR BAETJER:

Yes, it was cut down.

BY DOCTOR MIDER:

There were some more pathological changes other than the neoplasm in these animals?

BY DOCTOR BAETJER:

Yes, there were more. The inhalation mice showed very marked difference. The inhalation rats did show a higher degree of inflammatory reaction. In the intra-tracheal, there, of course, we were using definitely measured doses to put in, and there we had gotten marked fibrosis with atelectasis in the mice when we injected the mixed dust. That, at the point where it was injected, so that we knew we were getting it down in, but - and those animals, the weight gain was not too different from the controls.

BY DOCTOR RHOADS:

Now, I'm afraid I missed your group three of mice, berium chromate animals.

BY DOCTOR BAETJER:

They showed no difference at all, except they were slightly - slight irritation, slightly more, but not, statisticians would never consider it to matter.

BY DOCTOR RHOADS:

Doctor Lanza, could you comment on this work?

BY DOCTOR LANZA:

No.

BY DOCTOR RHOADS:

The paper is open for general discussion.

BY DOCTOR KUSCHNER:

Doctor Baetjer, in your experiments, the lesions comparable to nasal perforations, nasal sseptum, you mean

that is almost universal in the human experience?

BY DOCTOR BAETJER:

There was nothing in the rats or mice, we are now doing rabbits, and we are now getting nasal perforation of the rabbits, we didn't have it in the mice. We had no evidence of any malignancies there.

BY DOCTOR HERMAN:

There was one other thing, there was a recent case report in which liver damage was alleged in a - in a worker using chromate, I think in a man employed in a plating industry, and it's also been alleged, I think, that renal lesions have been caught. Could you comment on that on either liver or kidney lesions?

BY DOCTOR BAETJER:

We have not studied -- our pathologist - as a matter of fact, did had no pathologist on the project for a year, because he has been ill, so that the other features have not been studied. We have them, so I would not like to comment. In the literature - I have never found any description in the literature that chromium causes any liver damage. That does not seem to be characteristic. There are many reports in the literature, of animal experiments in which the chromates have been injected and which have caused kidney damage. Doctor Riegler has done some of those. Doctor Hueper has done some; he can perhaps answer

your question better than I.

BY DOCTOR HUEPER:

With reference to liver damages, refer to the Journal of the American Medical Association, I think it's published in New York, Liver Damage after Exposure to Chromate.

BY DOCTOR BAETJER:

I'd like to say with regard to that paper, my comment about it, if it's worth anything, and it probably isn't, is that I thought it was an extremely weak case for chromate. They had one case of liver damage in a chromium industry, and, therefore, they say chromium causes it. They had four persons working near there that had slight changes in the liver test, but no other changes whatsoever. It seemed to me that paper was extremely poor.

BY DOCTOR KUSCHNEAR:

I should like to refer back to a statement Doctor Baetjer made in reference to Doctor Lanza's paper. She stated that the pathology in these cases of chromate carcinoma was identical to that found in the garden variety carcinoma. I think, to pathologists and clinicians, that the lung carcinoma is unessential in a great many cases, and with Lindberg's study on the genesis of carcinoma, there have been many changes in many portions of the bronchial epithelium. Some people, indeed, assume that this unicentral

character is an argument against the importance of inhaled carcinogens. I don't think that's necessarily so, but I would like to know if in these chromate carcinoma, there are multicentric tumors or is there metaplasia, or perhaps carcinoma in sight, to be reassuring that in this type which seems to be so definitely related, if there were demonstrations of effects on the whole bronchial tree.

BY DOCTOR BAETJER:

As far as I know, Doctor Hueper can probably answer this question; from the case histories that I have read, they were localized. There were sixty-eight percent in the right bronchial tree, and the rest in my series, and I think the German series was sixty percent in the right bronchial tree. The - all these cases have been localized and are not distributed throughout the bronchial tree.

BY DOCTOR KUSCHNER:

Were there extensive studies of the bronchial tree?

BY DOCTOR BAETJER:

No, there were not, as far as I know.

BY DOCTOR RHOADS:

Can anyone else answer the question, very extensive studies being made of the changes in the bronchial necrosis in general; have you data on that point, Doctor Hueper?

BY DOCTOR HUEPER:

I couldn't tell you that.

BY DOCTOR RHOADS:

Does anyone have information? I think a very important point has been raised here certainly.

BY DOCTOR BAETJER:

I don't think any results have been published on the results that I have seen.

BY DOCTOR MEREWETHER:

Could I ask, sir, whether Doctor Beatjer has any cases of isolated carcinoma in the lower lobes? Professor Gross, when I saw him about seven years ago, said he had had a few cases or one or more anyway, that had nothing to do with the bronchi, the node bronchi.

BY DOCTOR BAETJER:

All of my cases were concerned with definitely bronchogenic, except one in which we had a pleural cancer in addition to the bronchogenic one, and it was of a different type, so it was not apparently related to it. We had some in the lower lobe, but as far as I know, they were all related to the larger bronchi. At least, so according to the pathology report that appears on the history.

BY DOCTOR RHOADS:

Are there further questions on the chome question? (No response). If not, we will pass to the last paper, that of our host, Doctor Vorwald, who will discuss his

BY DOCTOR REHADS:

Are there further questions on the chrome questions?

(No response). If not, we will pass to the last paper, that of our host, Doctor Vorwald, who will discuss his

studies on pulmonary cancer in the experimental exposure to beryllium.

BY DOCTOR VORWALD:

I'm sorry to say the report I'm about to give emanates from the work of my colleagues, particularly Doctor Pratt, and Mr. Durkan, Mr. Delehant and Mr. Urban of the Saranac Laboratory. Also, the second remark is that the hour is late and as host to this conference, I'm sure that they will agree with me when I cut my paper drastically, so that I will not take longer than ten minutes, if that. Third, I am not going to bow to those who champion the human experimentation versus animal experimentation because these observations are purely accidental. They weren't planned. The animals performed the experiment for us, and we have relatively little to do with it, except to place them in an environment for other purposes.

Briefly then, the report involves, this pilot or tentative report involves an experiment where we were exposing large groups of white rats to inhalation of atmospheres contaminated with beryllium in the form of pure beryllium sulphate, in one instance, and in the form of pure beryllium oxide in another. The animals, the white rats, and I call attention to that, that these animals are not mice, were bred in our own stock and they come originally or are descendants about 1935 from the Worcester Institute,

and no attempt has been made to inbreed or to select the strain and the strain is subject to lymphosarcoma at a low incidence rate which is not precisely known by us, because large enough groups of animals have never been kept in a duration of life experiment.

All rats under this observation were fed ad lib with a standard diet of prepared dog chow and water. The rats were exposed to pure beryllium sulphate in an atmospheric concentration of 0.011 milligrams per cubic foot of air. The rats were kept there under exposure for usually six and a half to seven hours daily for five days a week and the animals which I will report on, one of the observations made or the observations which I will report upon are made upon animals that have had that exposure continuously for approximately thirteen months.

The first slide, Jim. And, I will, rather than to show you all of the epithelial changes that have gone on in the bronchial tree of these white rats during the course of their exposure, namely, at one month, at three months, six months, nine months, ten months and twelve months, I take you immediately to the fourteen month animals. At that time, there were only four white rats left in the experiment in the large group of - well, in the group of fifteen female white rats being exposed to beryllium sulphate in the concentrations which I have given. At thirteen and three quarters

months, it was observed that one animal was ill, and so as is our custom, that animal was sacrificed rather than to allow it to die, and we, on killing the animal, we observed a very strange lesion which we had never heretofore observed in our rat colony, and here is the gross lung of that rat, and we see that there are para-broncholar foci of reaction to the beryllium that has become beryllium sulphate, that has become localized in those areas in the lung and in addition, we see also rather consolidated areas, and here is one, and we can pick some out.

Here is another, and in this area here, and also this large tumor mass at this point. That is one area.

The next slide. And, the second animal, now, subsequently then, having made the observation, killed all three remaining animals, remaining in that particular experiment, and I shall now show you the lungs of the three remaining animals and we have here the lung of a rat at fourteen months of exposure, the gross lung. We see the tumor localized in the apex. We can see the small lesion there. We can see it in the lugs, we can see a small lesion at that point. We can see a small lesion here and here and here. I merely take pains in pointing them out to show that the tumors are multiple within the lung tissue, and here is a larger tumor which was one - on one of the lobes which is not included in this section, and we see it - there

is a serial section and we see the tumor in the lowest portion of that lobe. This is the mark where I have taken the photomicrograph.

The next slide. The third and again, the fourth animal. Mind you, all four animals having been exposed for fourteen months to beryllium sulphate, 0.11 milligrams per cubic foot of air. At twelve months, we do see pronounced epithelial hyperplasia and I haven't bothered to show you those sections, but we did not believe at the time nor do we believe now that that epithelial hyperplasia had become cancerous but at fourteen months, it has. That again is another gross section.

We show you one of the larger tumors. Here is another tumor implanted on the pleura. Here is another tumor implanted on the pleura. Here is three tumors implanted on the pleura and we question whether this tumor is not infiltrated and replaced by the tracheo-bronchial lymph node which ordinarily occupies the proliferation of the lobe at this point. We're not sure about that, but we believe it has happened, because there is no evidence of lymph node tissue at this site which is common to both human subjects and in animal subjects, namely the trifurcation of the bronchi.

I will proceed briefly to show you the microscopic of these sections. Now, this then is a section photomicro-

graph from the major bronchial tree, and I call your attention to the epithelium lining the major bronchial tree, columnar in type, slightly hyperplastic and at some points the submucosal lymphoid tissue has apparently broken through which may be a normal observation, and here we see that lymphoid tissue exposed. I won't discuss that further. There is a problem here which I do not think pertains at the moment, to our immediate problem.

The next slide. And, as we go further down into the bronchial tree, we do find foci where a cross-section of a respiratory bronchial made up of epithelial cells, thick at the mucosa, evident hyperplasia, but in addition, we also find the adjacent alveolar spaces which are distorted by reason of the fibrous tissue or the cellular reaction of the alveolar walls, and we find those alveolar spaces lying with a very well defined epithelium which in instances is columnar, but in most instances is of the flat colloidal type. This is common, a common finding in the lung, subject to chronic diseases which give rise especially to fibrosis.

It is seen in human, in the lungs of human subjects, as well as it is seen in the lungs of experimental animals. Here is the type of lesion which we think simulates the lesion seen in human subjects, which characterizes that lesion due to the composition of beryllium. Made up of large mono-nuclear cells and very exact. I only

point that out in passing. That is the lesion which we are attempting to reproduce and we believe that we have reproduced it in these animal subjects, the rats, but not in any other animal have we produced a reaction. We have produced a reaction after intravenous injection in the rabbits.

Doctor Gardner first showed this with zinc beryllium sulphate, a BeO content of 13.8 percent and subsequent to that time, we have carried on a large number of experiments with various beryllium compounds, and we have reproduced the similar type of osteogenic sarcoma with metastases to the lungs with various beryllium compounds.

In passing, I might say that we were unsuccessful in producing these lesions with beryllium stearate, with beryllium carbonate, with - I want to get the list here so beryllium stearate, beryllium carbonate was too toxic, it wasn't tolerated by the rabbits intravenously. Beryllium hydroxide and beryllium metal, we have not succeeded in reproducing this tumor in the bones of rabbits, but we have done so with zinc beryllium silicate, a 13.6 percent BeO, 18 percent BeO, 2.3 percent BeO. We have done the same thing with pure BeO delivered intravenously in rabbits, in lower doses, the lowest dose was 650 milligrams. The same thing was accomplished with beryllium phosphate, in a dose of a hundred and two, 102.2 milligrams. I say that only because some of you are interested in the dosage factor.

So that this epithelial hyperplasia lining alveolar spaces is a common finding, and I am not going to enter the debate at the moment as to whether this re-epithelialization represents a hyperplasia of epithelial to epithelium which normally lines the alveolar space or whether it represents growth of epithelium from the adjoining respiratory passage which we know is lined with epithelia.

This then is the slide of the tumors. Now, and it is a deposit of all these four animals, and we find here two different types of tumor, one which is distinctly adenoidus in type and one part of the same tumor which is more alveolar in type, tending to reproduce at times at the alveolar architecture. Well, let us go on, and examine some of these tumors.

And we'll concentrate, first, upon the adenomatous type of tumor, that is the adeno-carcinoma seen in these lines and here we have a high-power magnification. It demonstrates the heterogeneous growth of this tumor tissue, a tendency to form spaces adenomatous in type. Certainly, there are many cells which are antiplastic, they are large, they are hypertraumatic. We can discover here many hypertrophic figures.

The next slide, and taking you to a higher magnification because my attempt here will be to show that this cellular change, this tumor is, we believe, a truly

malignant, and it has been submitted to other pathologists who have reviewed the section. Again, a higher magnification, you can see the cell structure.

The next slide, and still a higher magnification to show you the differences in size and staining capacity of these cells. No reproduction really of a specific pulmonary tissue growing in the tissue and also producing implants or metastases go to the pleural surface and metastases also going to the tracheo-bronchial lymph node.

The next slide, as to the alveolar structure, the alveolar type, indeed, this has been called by some in contrast to the respiratory bronchio-carcinoma, the alveolar type carcinoma and here we see a reproduction of the pulmonary alveolae lined with the malignant cells. And here a higher magnification to show you the character of that cellular growth.

The next slide, and still a higher magnification of this alveolar type of structure and we see a multitude of cells. This is now lining an alveolar space and hyper-traumatic, there is not myotic figure, except possibly that may be one, I don't remember the particular detail of this section.

The next slide is a section from the pulmonary implant tumor, and here it is tending to reproduce the major tumor in the - within the pulmonary branch. The next

slide, and here is the high powered magnification of that tumor on the implant, the megalocytic lesion, and here we have obvious metastases going on, extreme activity. Again note the difference in size and shape of the cells and the hypertraumatic richness of the nuclei, and the large size of the nucleoli, all of which is characteristic for a true cancer.

The next slide, and this then is the area of the tracheo-bronchial lymph node, this is the large mass bound at the trifurcation of the trachea and these merely represent serial sections because we were looking for differences in the growth of this tumor.

The next slide shows you now the tumor which has apparently replaced the metastases too and replaced the entire tracheo-bronchial lymph node and here again we see the character of the cell structure reproducing the primary tumors occurring within the pulmonary trachea, and this tumor is highly vascularized in the tracheo-bronchial lymph node.

With that then, we have now with BeO, we have subjected animals to BeO, pure beryllium oxide exposure, the concentration there was approximately 12 milligrams per cubic foot of air and the animals at that experiment were carried along for only eleven and one third months, not long enough to reproduce the same type of tumor as we have observed

with beryllium sulphate.

However, at this time, we saw similar epithelial hyperplasia in the bronchi as demonstrated in rats under exposure to beryllium sulphate, but we can point out that intra-tracheal injection of beryllium oxide, we have reproduced the same kind of tumor on intra-tracheal injection of beryllium oxide as we have with the beryllium sulphate, and also the beryllium sulphate on intra-tracheal injection, those animals have gone for only eight months, but the animal killed at eight months also shows a similar epithelial hyperplasia and lining of the alveolar spaces, and we suspect that these animals with longer periods of time, when they approach the fourteen month period, will show comparable changes.

So, with that then, we believe that we have produced in the white rat pulmonary cancer of the adenocarcinomatous type, with metastases to the pleural surface and to the tracheo-bronchial lymph node. We have not as yet observed distant metastases. This, as I mentioned, is only a pilot experiment. The observation is only tentative. It involves only four animals, but all four animals, all the animals remaining in that inhalation experiment, so we have set up a very large experiment in order to reproduce and to validate these observations.

We have abandoned the sacrificing period so that

Chapter Twenty - one

Pulmonary Cancer in Experimental Exposures to Beryllium

Arthur J. Vorwald, M.D.

The report I'm about to give.....

.....

.....cancer in human subjects.

Discussion

MR. McCORMACK: What concentration of beryllium was used?

DR. VORWALD: The concentration was 0.01 milligrams of BeSO_4 per cubic foot of air.

DR. BAETJER: What dose was used for the intratracheal experiments?

DR. VORWALD: In the intratracheal experiments we injected into the rats 0.25 cc. of a 5 per cent suspension of the dust, once a week for 3 weeks. The total amount of dust injected was 37.5 mg.

DR. HARDY: In relation to life span, what age of a man would be comparable to the age of a 14 months old rat?

DR. VORWALD: I suppose that 14 months of exposure in the rat might be equivalent to perhaps 25 years of exposure in human beings. Our rats seldom live longer than two years, so I believe that at 14 months they are approaching the age period when they may develop pulmonary cancer.

at the conclusion of the exposure, we will have an adequate number of animals which will give validity to our observations. I should like to point out here also that this is a pilot experiment, that this has been produced in white rats, that this does not mean that beryllium will produce pulmonary cancer in human subjects. Thank you.

(Applause).

BY DOCTOR MIDER:

Doctor Rhoads has delegates his authority to me, finding it necessary to go back to New York. It would seem unequivocal that Doctor Vorwald and his colleagues have produced real lung neoplasms in an animal with which all of us are familiar, and a species in which the spontaneous occurrence of neoplasms seems to be excessively rare. He and his colleagues are to be congratulated for this most timely report. May we have some discussion? Are there any questions which someone would like to ask?

BY DOCTOR McCORMICK:

Would you repeat again, Doctor Vorwald, the concentration of beryllium used?

BY DOCTOR VORWALD:

0.1 per cubic milligram of air.

BY DOCTOR BAETJER:

May I ask about the intra-tracheal?

BY DOCTOR VORWALD:

The intra-tracheal was an injection over a period of four weeks consisting totally of 2.5 milligrams or 2.5 c.c.'s of a five percent suspension.

BY DOCTOR MIDER:

Any other questions?

BY DOCTOR VORWALD:

I will check that, Doctor Bastjer, I want to be sure of that, I have it here.

BY DOCTOR MIDER:

Are there further questions?

BY DOCTOR HARDY:

Ignore a clinician's ignorance for me to ask how old in a man's life a fourteen month old rat is, how long do rats live?

BY DOCTOR VORWALD:

Our rats seldom live beyond two years, so we believe this animal at fourteen months, and that may be a factor that they're approaching, this age period, when they may develop pulmonary cancer.

BY DOCTOR HARDY:

I was trying to translate it into the number of years that humans have carried a body burden of beryllium.

BY DOCTOR VORWALD:

Well, I suppose fourteen months in a rat, to that exposure, might be equivalent to twenty-five years in a human.

BY DOCTOR MIDER:

Any further questions?

(No response).

If not, this symposium will adjourn to 9:00 A. M.
tomorrow.

(Adjourned at 5:45 P. M.)

DR. VORWALD: We know there is a species difference; for example, collagen caused by the pulmonary deposition of free silica develops in a shorter time in the white rat than in the guinea pig or rabbit. I have no evidence, however, that the colored race responds with more collagen, under the same conditions of exposure, than does the white race.

DR. APNER: Isn't a keloid the same histologically as fibrous tissue ~~spatters~~ appears in the lung following exposure to silica and, if it is, shouldn't the response to X-ray therapy be the same?

DR. VORWALD: Histologically, the architecture of the keloid is different from that of the silicotic nodule; basically, both are collagen formed in the same way. I do not know whether collagen in the lung would be as amenable to X-ray therapy as the keloid might be.

DR. FRIEDMAN: Half of our population in the coal mines of Alabama is colored and the other half is white. We have been unable to detect any racial difference in the response to the dust, either in the roentgenogram or at the autopsy table; one can find just as much fibrous tissue in the white man as in the colored man. We examined one negro who had massive keloids but his bone specimen gave no indication of what his skin showed in keloid formation; there was no correlation between the two.

The mortality rate of colored miners that work underground is greater than that of the white miners but this difference is attributed to the better living conditions of the white people.

DR. BASTJER: Dr. Fletcher, is there any difference ^{between} in the tuberculosis rate of the general community in the English mining area to which you referred, ^{and} ~~in~~ ^{tuberculosis} ~~comparison with~~ the rate for the Welsh mining area?

Discussion

R. MEHEWETHER: I am greatly honored.....
.....
.....to this factor if it is material.

DR. RHOADS: Dr. Lynch, was there no correlation in your series between the extent of asbestosis and the occurrence of neoplastic change?

DR. LYNCH: The three cases of carcinoma in our series occurred in advanced cases of asbestosis, not in minor conditions where a diagnosis is made merely because asbestosis might be present.

DR. LANZA: I find it difficult to believe that the cancer incidence among asbestos workers in the United States could be anywhere near as high as it appears to be in England. We may have missed some cancer cases but I do not believe we could have missed so many if they had occurred to the same extent they apparently do in England. It is true, of course, that as soon as the harmful effects of asbestos were recognized by American industry a program of cleaning up the plants was instituted, and although all asbestos plants in the country have not attained perfection in industrial hygiene, great strides have been made, particularly in the plant in Charlotte, Dr. Lynch's city. Such a

^{Wise}
~~good job has been done, and~~ the number of workers in the industry ~~is~~ so small,
^{it is obvious}
that we shall never have the clinical experience with asbestosis that we have
had with silicosis.

I used to hear frequently in Canada that the Canadian asbestos ^{is} ~~was~~ less
damaging than the kind used in England where the major part of the asbestos
used, I am informed by my English friends, is Rhodesian asbestos, a variety
quite dissimilar in structure ~~and formation~~ and physical characteristics to
the Canadian kind. ^{I am told that there is a difference in the Canadian varieties,}
^{between the asbestos mined at the town of}
^{Asbestos and the kind mined at}
^{for some time.}
Since experimental studies in the past few years have
shown that different types of silica may produce entirely dissimilar pathological
effects, I am beginning to wonder whether we have given sufficient attention to
differences in the type of asbestos in our attempt to explain variations in
the rate of asbestosis.

DR. SMITH: I had ^{an} ~~the~~ opportunity to visit Dr. Glynn shortly before his death
^{Tb} and ^{in England.} learn something about the material he had assembled. He had 17 cases in
which there was co-existent asbestosis and carcinoma of the lungs. In his
series there was no detectable relation between the degree of asbestosis and
the presence or absence of cancer ~~or between the degree of asbestosis and the~~
~~site of cancer.~~ His series came from a plant that used the Rhodesian blue
asbestos which, as Dr. Lanza pointed out, has certain characteristics that are
different from those of Canadian asbestos. The Rhodesian variety has a more
brittle fiber and, therefore, gives rise to more dust. I understand, however,
that some of the ^{British} ~~Great Britain~~ cases had been exposed to ~~the~~ Canadian asbestos.

~~While the effect of variation of circumstances on observed phenomena was~~
^{Earlier} ~~being discussed,~~ the question of ^{carcinogenicity.} ~~exposure to~~ nickel was brought up. Recently,
I visited Dr. Anon^y, formerly medical officer for the plant in Wales where the

nasal sinus tumors had been observed, and Dr. Morgan, who is the medical officer there at present. They stated that the men who developed the nasal sinus tumors had not been employed in the part of the plant where there might be exposure to nickel carbonyl, ~~and~~ ^B Both doctors were very reluctant, therefore, to believe that ~~the~~ nickel carbonyl ~~was~~ a factor in the nasal sinus tumors observed. They pointed out ~~also~~ ^{arsenical dust from} that the tumors which had occurred were in men exposed to the calciners and that cancer of the nasal sinus was not seen in men who were employed in the industry only after 1924, when the calciners were re-designed.

*See
but
not
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DR. MEREWETHER: I believe that Dr. Lanza has been misinformed about the proportion of the different kinds of asbestos used in England and about where they are used. The original cases of asbestosis there, which occurred 50 years ago, were caused by Canadian asbestos. In the early days of the asbestos trade, about 70 years ago, different kinds of asbestos may have been used but very quickly the industry concentrated on the white Canadian variety. Later on, Rhodesian asbestos - the blue kind from South Africa - and a third variety, amosite, which is also an iron silicate, came into use. Of these amosite is definitely the most brittle. It is brown in color and has fibers that are much shorter than the best Canadian spinning fibers or the best Rhodesian fibers. Amosite liberates considerable dust and is not extensively used today, but at the period when some of these cases of asbestosis occurred, a large amount of blue asbestos and of amosite was used.

Regarding the cancer from exposure to nickel we do not believe that nickel carbonyl has anything to do with the situation for the simple reason that nickel carbonyl is so toxic that ^{many} more persons would have been affected. Dr. Moore,

VORWALD COLL
BOX 89-91

VORWALD COLL
BOX 89-91

SEVENTH SARANAC SYMPOSIUM

THE SARANAC LABORATORY of the
Edward L. Trudeau Foundation,
Saranac Lake, New York

Thursday Session
September 25, 1952.

CLINICAL ASPECTS OF PNEUMOCONIOSIS

Chairman: William S. McCann, M. D.

Incidence of Clinically Manifest Diffuse
Obstructive Emphysema

Leonard J. Bristol M. D.

Discussion

Pulmonary Function Studies in Men Exposed for
Ten or More Years to Inhalation of Asbestos Fibers

Fernand Gregoire, M. D.

Discussion

Maximum Ability for Physical Work of Moderate
Duration Correlated with Various Tests of Pulmonary
Function

George W. Wright, M. D.

Discussion

Cardio-Circulatory Aspects of the Pneumoconioses

William S. McCann, M. D.

Discussion led by James J. Waring, M. D.

BY DOCTOR McCANN:

Ladies and Gentlemen, since the title of this morning's meeting is the Clinical Aspects of Pneumoconiosis, I should like to take the Chairman's privilege of making a few introductory remarks to this session.

There have been several points which have come up in the course of the recent meetings which seem to me worthy of comment. First of all, it was a great pleasure to learn

Should the papers by
Wright and Gregoire be given
before the discussion
of so, and chapter numbers are changed
EVER

PART FIVE

Clinical Aspects of Pneumoconiosis

Chapter Twenty - two

Introduction

William S. McCann, M.D.

There have been several points.....

.....to some extent.

Chapter Twenty - three

Incidence of Clinically - Manifest Diffuse Obstructive Emphysema

Leonard J. Bristol, M.D.

I should like to state.....

.....as in the dusty trades.

Discussion

DR. BENNETT: I Believe that the age group is an important consideration and should be included in the biostatistical data.

DR. HUGH-JONES: It is an exceedingly difficult job to find a suitable combination of things by which to define physiological values. Dr. Bristol and Dr. Wright are attempting to do something along this line, but there have always

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PART FIVE

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Chapter Twenty - two

Introduction

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Chapter Twenty - three

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DR. BENNETT: I believe that the age group is an important consideration and should be included in the biostatistical data.

DR. HUGH-JONES: It is very difficult to find a suitable combination of indices by which to define physiological values. Dr. Bristol and Dr. Wright are ably attempting to do something along this line. There have always been

difficulties in employing a series of tests to define a particular syndrome - in this case emphysema.

One of the difficulties is that although there may be a "high" degree of correlation of any one test with another, the error of prediction of the one from the other often ^{is} still very great. For example, I can draw *(on the board here)* the relationship between one function, such as a maximum voluntary ventilation and the timed vital capacity; there will be a certain scatter about the regression line which is the average relation between the two. If the correlation is "high", say about 0.7, the scatter is still such that when one is predicted from the other the limits of prediction are unhelpfully big.

The practical problem is to combine tests to give an index of maximum usefulness, and not to waste information in the prediction of one test from another, as if there were an absolute relation between them. Two tests usually only partially measure the same thing; it is in the residual variance about their regression relation that the specific quality measured by the one is rejected, along with test error, in prediction.

If one will admit that the clinician can define gross emphysema as compared with the normal - Dr. Bristol described that possibility - then one can take the different tests which describe the changes in emphysema (for example, maximum voluntary ventilation, residual capacity, carbon dioxide sensitivity, etc.) and find by a discriminant function what combination of them gives maximum differences between normal and grossly emphysematous groups of subjects. Such a function, might read, for example, something like "three times the M.V.V., plus four times an index of ventilatory inequality, less twice the arterial carbon dioxide". A new test added to the function will only increase the discrimination between the groups, if it is providing something not contributed by the tests already included. Such a discriminant function for "emphysema", for example, can then be applied to determine the degree of this characteristic in field results on cases of pneumoconiosis.

DR. WRIGHT: Dr. Hugh-Jones' suggestion is a good one. We attempted to get at that to some extent by a full-time correlation procedure, as explained in my paper (Chapter 24). Possibly Dr. Hugh-Jones' procedure is a little different and is one with which I am not familiar.

DR. FLETCHER: Regarding fibrosis and emphysema I should like to mention that in coal miners fibrosis is commonly but not always associated with emphysema.

Will you tell us, Dr. Wright what percentage of the total population covered by the studies refused to take the tests?

have difficulties in employing a series of tests to define a particular syndrome - in this case emphysema. One of the difficulties is that although there may be a "high" degree of correlation of any one test ^{with another,} the error of prediction is, unfortunately, very great. If a relationship between one function, such as a maximum voluntary ventilation, and the time of the vital capacity, there will be a certain scatter about the regression line. If the correlation is low or even as high as 0.7, when one is predicted from the other, there will be a scatter about this line, and to overcome this difficulty we have used what is statistically a discriminate function, that is, we combined a series of tests to maximize a difficulty or difference.

If one will admit that the clinician can define gross emphysema as compared with the normal - Dr. Bristol described that possibility - then one can take the different tests, those giving for example maximum ventilation, residual capacity, and CO₂, or tests predicted from them, and find what combination.....
....(text not clear).....and obtain a statistical function which maximizes the change from normal to abnormal.....(text not clear). You can get a discriminate function which gives the maximum difference in normal values and in values for extreme emphysema and then apply that function in the field to these other things, using the simple predicted tests.

DR. WRIGHT: Dr. Hugh-Jones' suggestion is a good one. We attempted to get at that to some extent by a full-time correlation procedure, as explained in my paper (Chapter 24). Possibly Dr. Hugh-Jones' procedure is a little different and is one with which I am not familiar.

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Will you tell us, Dr. Wright, what percentage of the total population covered by the studies refused to take the tests?

DR. WRIGHT: I cannot say precisely how many refused because I'm not sure that a complete record of the refusals was kept.

DR. HUEPER: What was the labor turn-over in the various groups tested? In dusty occupations there is generally a higher turn-over and, therefore, a shorter exposure time.

DR. WRIGHT: Data regarding the labor turn-over is being reviewed but is not yet in final form. There were a large number of men - possibly even the majority of those in the dusty occupations - that had been employed for 20, 25, 30 years.

Dr. Bristol pointed out that his paper was a preliminary report; in the final report more information about such subjects as dust exposure and labor turn-over will be given. The age distribution ranged from approximately 25 to 70 years, and care was taken in choosing the control group to have a similar age distribution.

When asking for volunteers from the dusty trades we obtained all of the people sought except 2 persons in the foundry and 8 in the porcelain operation. Those 10 persons were away on holidays; there were no refusals. I do not believe that our data was seriously impaired by the absence of those 10 individuals. A few persons in the tool and dye and punch press operations refused, but there were not many in number.

DR. BRISTOL: Would you comment about the incidence of emphysema in conglomerate silicosis?

DR. WRIGHT: In regard to emphysema being a complication of conglomerate disease it is our experience that in the vast majority of cases of conglomerate silicosis there is demonstrable evidence of emphysema. In one very

striking case, about which I published a report, there was no emphysema.

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DR. FRIEDMAN: Dr. Wright, how many in the dusty-trade groups you studied had radiologic evidence of pulmonary disease and how many were disabled according to ^{present} standards of pulmonary function studies?

Secret
DR. WRIGHT: Some members of the group had radiologic evidence - the exact number will be noted in the completed report - but other methods of study were not carried out on these individuals.

Secret
DR. FRIEDMAN: You may have been dealing with places where the improvement of dust conditions had already been accomplished. Do you know how many years these men worked in hazardous concentrations of dust and how many years they did not?

Secret
DR. WRIGHT: Yes, we have that information. It will be included in the final report.

Chapter Twenty-five

Pulmonary Function Studies in Men Exposed for Ten or More Years to
Inhalation of Asbestos Fibers

Fernand Gregoire, M.D.

(Neither transcript nor paper on file)

Out

Discussion

DR. HUGH-JONES: Dr. Wright offered a suggested definition, shall we say, of measuring a man's overall capacity for work. Taylor and Briggs did what they called the crest load, the maximum work that people can do steadily, and other people have done the maximum ability to work as measured by the oxygen consumption, used also by Dr. Wright. But one thing worries me: Although two persons may have very different powers to do their maximum effort, does that fact necessarily limit them at the ordinary work they do every day? Does Dr. Wright have evidence to show that the performance at sub-maximal loads is necessarily worse for people who show a falling-off of their maximum ability? It doesn't seem to me that such a situation must necessarily follow. For

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Dr. Wright's chapter

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See
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Hugh Jones
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Slide, 1941, 1942, 1943

BY DOCTOR MC CANN:

Ladies and Gentlemen, since the title of this morning's meeting is the Clinical Aspects of Pneumoconiosis, I should like to take the Chairman's privilege of making a few introductory remarks to this session.

There have been several points which have come up in the course of the recent meetings which seem to me worthy of comment. First of all, it was a great pleasure to learn

Chairman's Remarks

see corrections

Thursday, September 25, 1952

Seventh Silicosis Conference

Clinical Aspects of Pneumoconiosis - W.S. McCann

It was a pleasure to learn from Dr. Philip Hugh-Jones that there is so much validity to the clinical estimation of disability. He was, of course, speaking of British Clinicians. I wish I could be sure that his remarks applied equally to us here in America, where, it seems to me clinical medicine has fallen to a low estate, due to our national penchant for gadgeteering and for simon-pure specialization.

This leads me to address myself to answer the question - which naturally arises in your mind - what is so wrong about specialization? The answer is simple - as one learns "more and more about less and less" he tends to become unable to see his patient as a whole against the background of his whole environment. This is the very essence of understanding the nature and meaning of disease. As one becomes more and more specialized he becomes the very antithesis of the true clinician.

Let us attempt a definition of disease.

Diseases may be recognized and classified by:

- (1) Their symptoms and signs; (2) Their causes; (3) The nature of the pathological processes underlying them; (4) The character of the environmental setting in which they occur; and (5) The personality or "constitution" of the patient who suffers "dis-ease."

These are the pigments with which the pictures of disease - nosography - are painted.

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To this end aggregations of cells, which we call organs or systems of organs, have specialized functions in maintaining this constancy - this homeostasis.

These functions are integrated and regulated by the mediation of the nervous system, central, peripheral, and visceral; and by the balance in hormonal activity, which determine the character and constitution of the person or as we say, individual personality.

Each individual must adapt himself to his environment, not only that which is immediate, or local, but the entire environment - physical, climatic, religious, moral - every influence with which each person must strive for survival in peace and comfort or health.

When environmental factors cause stress - the strain of the individual to adapt to it produces symptoms and signs of "dis-ease."

Normal individuals develop symptoms and signs only when the stress is unusually great.

Abnormal individuals, whose adaptive functions are limited or damaged by pathological processes, show strain when the stress is not unusually large or even when it is less than usual.

Disease may occur then in both normal and abnormal persons depending upon the degree of stress relative to the capacity to adapt.

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As William Harvey said - "There are two kinds of death - failure from a lack and suffocation by an excess."

With this preamble I will now venture to suggest a definition of the pneumoconioses. They are a group of diseases characterized by shortness of breath and diminished capacity for exertion and the performance of mechanical work; caused by the prolonged and repeated inhalation of particles of substances, which in sufficient amounts are capable of producing morbid anatomical changes in the lungs and circulatory apparatus, such as fibrosis, emphysema, lymphatic obstruction, pulmonic arteriosclerosis, right heart hypertrophy, adhesive pleuritis, etc., in varying degrees and in varying permutations and combinations. They are classified by the character of the dust, by the nature of the occupational environment (mine, foundry, quarry, sandblasting, etc.)

In evaluating the degree of disability resulting from these diseases one must consider the constitution of the whole personality of the individual patient, including his motivation and his capacity to compensate for injury in one organ system by improved use of another. It must also take into account organic impairments which are not the result of occupational stress, such as age, habits, nutrition, inheritance, and environmental stresses outside the occupation. This involves a thorough general diagnostic study.

In closing these remarks, I would like to comment on the physical diagnosis of emphysema. I have heard it said that there is no pathognomonic sign of emphysema but I do not believe this. I think there is one sign; and that has to do with auscultation. The breath sounds in emphysema are of low intensity and pitch, and the expiration is of longer duration than inspiration. The use of a stethoscope is a

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The methods of science are like the old three decker, which took a watch to steer her and a week to shorten sail. They are applicable to the study of diseases, but much less applicable to the study of patients.

One final word about the teaching of clinical medicine. Some one wisely said that if teaching consisted only of imparting factual knowledge, there would have been no use for universities and professors after the discovery of printing. One would then need only libraries. However, the great teachers are those who can endow facts with their

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from Doctor Philip Hugh-Jones that there is so much validity in the clinical estimation of disability. He was, of course, speaking of clinicians. I wish I could be sure that his remarks applied equally to us here in America, where, it seems to me clinical medicine has fallen to a lower state due to our national penchant for gadgeteering and simon-pure specialization.

Naturally the question arises in your mind as to why I think there's something wrong about specialization. The answer to this is simple. As one learns more and more about less and less, he tends to become unable to see his patient as a whole person against his whole environmental background. This, it seems to me, is the very essence of understanding the nature and meaning of disease and as one becomes more and more specialized, he becomes the very antithesis of the clinician.

Now, the other day, the question came up of trying to make a definition of disease. I made a very lame attempt at it, and I should like to devote a moment or two to an attempt to define disease. Diseases may be recognized or classified by certain criteria. First, there are simple - there are symptoms and signs; second, their causes, third the nature of the pathological processes underlying them, fourth, the character of the environmental setting in which they occur, and fifth, the personality or constitution of

the patient who suffers disease. These are the pigments with which the pictures of disease are painted.

Tracing the history of nosography, Sydenham said: "Symptomata se habent ad morbum ut folia et fructus ad plantam." Symptoms are to disease as the leaves and fruits are to plants, and following Linnaeus, he tried to classify them.

Auenbrugger, Laennec, Stokes, Corrigan, Graves, Bright, Addison, Louis, Andral, Brettoneau, all related signs to morbid anatomical changes. Pasteur and Koch began the concept of specific causes. People like William Harvey, Magendie, Claude Bernard laid the foundation for pathological physiology.

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sufficient amounts, are capable of producing morbid anatomical changes in the lungs and circulatory apparatus, such as fibrosis, emphysema, lymphatic obstruction, pulmonary or pulmonic arterial sclerosis, hyper... right heart hypertrophy, adhesive pleuritis, and so forth, in varying degrees and in varying permutations and combinations. They are classified by the character of the dust, by the nature of the occupational environment, such as mine, foundry, quarry, sandblasting, and so forth, and perhaps within these categories, local designations such as Welch coal mines or Lancashire coal mines or Pennsylvania hard coal area, and so forth.

In evaluating the degree of disability resulting from these diseases, one must consider the constitution of the whole personality of the individual patient including his motivation and his capacity to compensate for injury in one organ system by improved use of another. It must also take into account organic impairments which are not the result of occupational stress, such as age, habits, nutrition, inheritance and environmental stresses outside the occupation. This involves a thorough general diagnostic study.

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do not believe this. I think there is one sign and that has to do with auscultation. The breath sounds in emphysema are low intensity and pitch and the expiration is of longer duration than the inspiration. Now, in appreciating these characteristics of the breath sounds in emphysema, I believe that a stethoscope is a handicap. A stethoscope is, after all, a good deal like the receiving set of a certain radio. It will separate bands of frequencies of sound and very often certain bands are cut out or blocked by the stethoscope. I, therefore, think that one should routinely listen to the chest with the ear directly, in order to appreciate this and to pay attention to intensity, pitch and duration of sound. In this manner, the presence of emphysema may be detected when the X-ray picture is so full of density that it is obscured.

Now, the role of the clinician in the study of disease is like that of man trying to put together the pieces of a picture cut by a jig-saw. He is first to examine the pieces in order to guess at the nature of the assembled picture. The clinician must guess, he must speculate, he must postulate as to the final pattern if he is to put the pieces together quickly, quickly enough to be of any use.

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I was moved to make this last remark because, in my own presentation, I have allowed my imagination to work on the facts to some extent. (Applause).

Now, I think I have taxed your patience over long.

We will proceed to the first paper on the program by Doctor Leonard Bristol, on the incidence of clinically manifest diffuse obstructive emphysema. Doctor Bristol.

BY DOCTOR BRISTOL:

Doctor McCann, Ladies and Gentlemen of the Symposium: I should like to state at the outset that this is a preliminary report. It is a combined study of the Departments of Physiology, the Radiology of the Trudeau Foundation and the physiological data in this paper has been compiled by Doctor George Wright and his staff. I should like to have it clearly understood that my part in this work was that of a radiologist.

The word emphysema is derived from the Greek and literally means a blowing. Strictly speaking, it is defined as a swelling produced by gas or air diffused in a cellular tissue. It is a term of broad general meaning and, therefore, when it is employed for descriptive purposes, one must define clearly what is meant by its uses.

The clinical diagnosis of chronic pulmonary emphysema is not simple to make. No physician should have difficulty in establishing a diagnosis of far advanced emphysema, but it requires the most keen clinical insight to diagnose correctly a mild to moderate emphysema. A large proportion of moderate emphysemas are overlooked while many cases so diagnoses are not confirmed by pathologic study.

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In a recent publication, Fernon states: There is no consistent relationship between the clinical severity diffused by lateral pulmonary emphysema and the extent of the process seen in necropsy. This, in general, has been our experience. Histologically, pulmonary emphysema is a condition in which the alveolae are distended, distorted and interrupted. Continued air sacs within a lobule may coalesce and adjacent lobules may fuse to form large air spaces. As a result of this, the total respiratory surface is reduced and the pulmonary capillary bed is diminished. The pulmonary elastic tissue is frightened and appears to be reduced in amount. It is surprising that one of the chief clinical and physiological features, namely, air with an obstruction, has not, with the exception of a recent paper by Spain and Amberson, been described by histologists.

The term emphysema as used in medicine is very ambiguous and as - is far too often employed in a very loose manner. Therefore, it is imperative that we think clearly as to what is meant when we use it. Several varieties of this condition are recognized. A simple but satisfactory classification has been devised by Rubin -- may I have the first slide, please -- as follows:

Obstructive emphysema caused by mechanical interference with respirators. The subdivisions be neath that, as you can see it, are chronic hypertrophic bulbous forms,

two, common obstructive emphysema caused by obstruction, in the thoracic change. The lungs expand to fill a barrel shaped chest, referred to as senile or postule emphysema. Interstitial emphysema condition characterized by tissue trauma of the lungs; for compensatory emphysema, a state of physical enlargement of the alveolae to meet anatomic readjustment and increased functional demands resulting from loss of tissue and, five, combined forms of emphysema, one component usually being predominant.

At this time, emphysema may develop suddenly or slowly and is usually bi-lateral and generalized. This condition may occur independently of any pulmonary disease, as a complication of long standing bronchial disease or may be associated with other diseases or malformations of the lungs or chest.

For purposes of discussion in this paper, we shall limit our consideration to diffuse obstructive emphysema for the chonric hypertrophic form, The salient clinical feature of which is airway obstruction. Whatever the obstructive agent, the mechanics of this condition are such that air entering the lung during inspiration is exepelled with difficulty during expiration, the difficulty in turn leads to a reduced maximal breathing capacity, a high residual air and in its advanced stages, to a retention of carbon dioxide in the lungs and hypoxia.

These physiological data can be recorded accurately in relation to an individual's total respiratory capacity. I should like to present a case of classic diffuse structural emphysema. This slide represents a film in full inspiration and full expiration, the production on your left being the inspiration microgram and the one on your right in full expiration.

I should like to direct your attention to the lack of rise of the diaphragms in the expiratory film and the failure of this patient to empty his lungs on expiration. The information to be gathered from this slide is that the patient has a low maximum breathing capacity and a higher residual air. We have predicted maximal breathing capacity of twenty-two liters per minute at determined thirty-one; residual air, predicted 1.36 and determined was 1.54.

The data on this slide shows that the patient has an elevated carbon dioxide content, an abnormally low hemoglobin saturation with .02. There is an elevated PCO_2 and a low PO_2 . These are evidences of markedly impaired ventilation of the blood.

Diffuse obstructive emphysema is known to be one of the complications of silicosis. Simple discreet nodular silicosis is seldom associated with clinically manifest diffuse obstructive emphysema. On the other hand, conglomerate silicosis is almost always complicated by some degree of

diffuse obstructive emphysema. We do not mean to imply that chronic pulmonary emphysema is never detected in an individual who has discreet nodular silicosis, but raise a question as to its underlying cause when the two are associated.

Is it directly related to the pulmonary deposition of quartz or is it entirely an independent process? Emphysema has been alleged to be a complication of asbestosis. This problem will be discussed by Doctor Gregoire. Emphysema is also alleged to be caused by the inhalation of coal dust. Emphysema has been observed in granular beryllium workers, but is by no means always present. Emphysema is also observed in persons exposed to all manner of inert dust and some go so far as to associate these inert dusts in a causal relationship.

Now, to establish a causal relationship between emphysema and the inhalation of any foreign material, requires the demonstration of a high presence of emphysema in those so exposed as in a similar group not so exposed.

We propose to explore the question of whether or not there is a higher incidence of emphysema in those exposed to the inhalation of inert dust than in those not so exposed, in an attempt to prove or disprove a causal relationship between such inert dust and the development of diffuse obstructive emphysema.

The first step in such a study is to discover the

relative incidence of emphysema in a dusty and non-dusty trade. As mentioned before, diffuse obstructive emphysema is difficult to diagnose by means of history or physical examination, except in advanced cases. Hence, it is imperative that we look for more refined methods.

In recent years, clinical physiologists have devised accurate techniques in the study of the normal and abnormal functions of the lungs. These various lung function studies offer a method of appraising pulmonary disabilities objectively. If we again refer back to our original case which, as you will recall, was that of a patient with diffuse obstructive emphysema, it will be seen that there was the presence of a low maximum breathing capacity. This test measures the maximum volume of air that can be breathed in a unit of time. The patient also had a high residual of air. This represents the amount of air remaining in the lungs following a maximal respiratory effort.

Also, there was a retention of carbon dioxide and hypoxia. Obviously, it is impractical to employ these methods in undertaking a study of a large series of individuals. Therefore, we have attempted to find a more practical procedure. The maximum breathing capacity is known to correlate highly with the volume of air that can be expelled in a unit time.

Gensler has shown that the maximum breathing

capacity correlates highly with the volume of air expired in one sector. In a similar study carried out in our physiology laboratory, Wright and his co-workers found that the volume of air expelled in eight tenths second following the beginning of the most rapid expiration possible, correlated with the maximum breathing capacity of .758 with a standard error of .750. It will appear from these studies that the air expelled in eight seconds during such a maneuver and hereafter referred to as E2, is to a limited extent a substitute for the maximum breathing capacity test in mass surveys.

May I have the next slide, please? It would appear logical that the enclosed area within the silhouette of the thoracic cage at full inspiration would bear a relationship to the total volume of air within the lung, that the silhouette of the thoracic cage at full expiration would bear a relationship to residual air. The possibility of using the ratio between these two areas is a substitute for the actual residual air total volume ratio, has occurred to several investigators.

On this slide, a-3 refers to the area at full expiration and A-1 to the area of full inspiration.

In 1933, Gertado and his co-workers showed that there was a significant correlation between residual air and total volume ratio and the area enclosed within the

thoracic porages on a film taken at full expiration, provided that - in a similar area on full inspiration. Five years later, in another communication, he stated that contrary to previous results, the correlation between the ratio, area at maximum expiration divided by area at maximum inspiration and the maximum residual air divided by total capacity, was low and insignificant, but no explanation of this statement was ever made.

May I have the next slide, please? Recently, George Wright has studied two hundred and thirty-four cases and showed a good correlation of the residual air total volume ratio with the area of radiological inspiration, area of radiologic inspiration, the co-efficient being .772 and with a standard error or .02, and the remainder of this paper, this radiologic ratio will be referred to as A-e, A-1.

This is the regression formula showing that high correlation of stock that Wright obtained in this study. You will notice in the right-hand corner is marked 134 cases but since this lamp slide was made, he has studied a hundred more, making a total of 234 cases. From this regression formula, we can see that there is a high correlation between the area expiration and area of inspiration, with the percentage of residual air.

Now, this lantern slide represents a full

expiration of a normal healthy male. The area of the film, the area of the film on your right is referred to as A-3 and the area of full inspiration on your left represents A-1. These measurements are determined by a clinimeter and you can see the tracings which have been made especially on the expiration film, to determine from these traces.

The work we wish to report on this morning represents a study of 713 men in the General Electric plant in Schenectady, by means of inspiration and expiration films and measurement of maximum expiratory velocity. The men studied made up three distinct groups, one, all but two of those currently employed in the iron and steel foundry, or 237 persons; all but eight of those currently employed in the porcelain division or 193 persons; workers from the tool and dye and punch press divisions, 229 persons; these last were chosen by random sampling, but were allowed to refuse if they did not want to participate in the study. Fifty more men from salary personnel were also studied.

Most of these have been employed more or less sporadically than the laboring group of the divisions mentioned. The non-dusted group, at the same age distribution as did the dusted groups. Each man had the following study: Roentgenograms were made on a four by five flat film, the films taken as follows: One, full inspiration, two, four tenths of a second after beginning of the most rapid

expiration possible; three, eight tenths of a second after the beginning of the most rapid expiration possible, and four, in full expiration.

We are not reporting on the four tenths or eight tenths seconds films at this time. All exposures were set at one tenth of a second, The kilowatts and high amperes being slightly over exposed for the film in that period.

The expired air was caught in a spirometer with two indicators, one of which was stopped at either four tenths or eight tenths second after expiration was begun. The other registered the full vital capacity. The volume of air blown into the spirometer during the four tenths or eight tenths second period is considered to be representative of maximum expiration velocity. The maximum breathing capacity was measured in 233 of the 713 men.

The following data have been calculated regarding the maximum expiratory capacity. The first three lines are representative of - define our symbols. E-1 equals the volume of air expired in four tenths of a second at the fastest expiration possible. E-2 represents the volume of air expired eight tenths of a second at the fastest expiration possible and E-3 equals the vital capacity at fastest expiration possible. RE equals the co-efficient of correlation.

Now, I'd like to draw your attention to this line

here, apparently E-2 correlates highly and best with the actual maximum breathing capacity. This is all the more significant when one realizes that we were studying a group of individuals who are almost normal from the standpoint of their maximum breathing capacity. As indicated by the fact that their mean maximum breathing capacity was 135 liters per minute and the median 140 liters per minute. If we had had a larger proportion of persons with a pathologically low maximum breathing capacity, we probably would have had an even higher correlation co-efficient.

If one would accept the contention that the E-2 is a fairly representative measure of the maximum breathing capacity especially for group studies, one can derive further information from the study. One hundred and six men had a maximum breathing capacity that fell within one standard deviation of their predicted normal figure, and a hundred and sixty fell within two standard deviations. The mean E-2 for the hundred and six men was 2.934 liters, and for the hundred and sixty men, it was 2.836 liters.

One may, thus, take the figure of 2.836 liters as the mean E-2 for a group of men who have normal maximum breathing capacity.

Of the entire group whose maximum breathing capacity was measured, 32 have values outside the range of normal, that is beyond two standard deviations on the low

side. The mean maximum breathing capacity of this low group was eighty-eight liters per minute and their mean E-2 is 2.198 liters, in contrast to the normal group. It is apparent that the E-2 will disclose the presence of a low maximum breathing capacity.

If one starts with the premise that those mean with a normal breathing - maximum breathing capacity, that is within two standard deviations of their predicted, comprise a group which is, as a whole, free of emphysema, then their mean E-2 plus two standard deviations and their mean A-3:A-1 ratio plus two standard deviations can serve as a control for comparison with other groups.

The mean maximum breathing capacity of 160 men, within two standard deviations of predicted, covering ninety-five percent of their group, is 139.97 liters, and is 97.17 percent of predicted values. The E-2 of this group is 2.836 with the standard deviation of .551.

Next slide, please. From this data, is it - it is seen that the mean E-2 of these 713 men compares well with the mean E-2 of the 160 men whose mean maximum breathing capacity was 97.17 percent of predicted values. The only exception was in the iron foundry group and this difference was only slight, the difference being .164 which is slightly more than twice the standard error of the difference.

So, therefore, there is just as many pathological

E-2's in all groups, and insofar as E-2 correlates highly with maximum breathing capacity, we can state that there are just as many people with low maximum breathing capacity in the non-dusty trades as there are in the dusty trades.

The next question we must ask ourselves is what is the percentage of men within the various groups who fell below two standard deviations of the mean E-2 of 2.836. Next slide, please. From this chart, we can see that there was one out of fifty-six in the steel foundry or 1.8 percent, five percent in the iron foundry, 31 percent in the porcelain division, 5.8 in tool and dye, 8.3 in punch press and 9.3 in salaried personnel. There were as many men in the non-dusty trades who fell below two standard deviations as there were in the dusty trades.

This demonstrates quite conclusively for the group studied, this, that there were as many men, if not more, in the non-dusted group who have a below normal E-2 as there are in the dusty occupations.

Next slide, please. The mean A-3:A-1, that is area of expiration over area of inspiration, from the radiological films, from the films of those men having a normal maximum breathing capacity, is .615 with a standard deviation of .058. Now, it is reasonable to assume from this information, that there is some increase in residual air in the foundry and porcelain workers, but not enough to cause

a reduction in the E-2, and by inference, the maximum breathing capacity. The number of men in this group who have a pathological A-3, A-1 ratio, in other words, more than two standard deviations from the men of normal mean, is demonstrated on the next slide.

The steel foundry, 7.9 percent; iron foundry, 5.8 percent; porcelain division, 5.9 percent; tool and dye, 1.8 percent, punch press, 5 percent; salaried personnel, 2 percent. Now, from this we can readily see that the number of men who have an increased residual air is slightly higher in the dusted versus the non-dusted groups.

Now, in conclusion, I should like to say that this has been an exploratory study made in an effort to obtain information as to the degree of emphysema present in certain occupations. The E-2 of all groups was approximately the same, and the A-3:A-1 ratio in the foundry and porcelain divisions departed only slightly from that in the group of men employed in the non-dusty trades. Because E-2 has a high correlation with maximum breathing capacity and the A-3: A:1 ratio correlates highly with residual air, it is easy to assume that these studies in mass survey will render reliable information as to the quantity of emphysema present in any particular group.

These methods failed to reveal any presence of significant change among the various groups studied. If

the pulmonary deposition of dust is the cause of an increase in the incidence of emphysema, then one would expect, from this investigation, to find it clearly demonstrable, the difference between the two groups studied, such a difference was not apparent.

In closing, I should like to reiterate that this is a preliminary report and that the information to date suggests that there is just as much emphysema in the non-dust trades as in the dusty trades. Thank you.

(Applause).

BY DOCTOR McCANN:

Doctor Bristol's paper is open for discussion.

BY DOCTOR BENNETT:

Doctor Bennett, New York State Workmen's Compensation Board. I believe the paper and the presentation of its biostatistical data should have had the age groups referred to, because this may well be the universal statistical computation, namely the study of emphysema in various trades. However, the age group is an important thing.

BY DOCTOR McCANN:

Are there any other comments? Doctor Hugh-Jones?

BY DOCTOR HUGH-JONES:

Mr. Chairman, I would like to congratulate Doctor Wright and Doctor Bristol on this - on their work. One of the very difficult things, indeed, is to find practicable

TABLE VI

MAXIMUM O₂ UPTAKE PER SQUARE METER OF BODY SURFACE CORRELATED WITH:

| | <u>No. Studied</u> | <u>"r"</u> | <u>"p"</u> |
|---|--------------------|------------|------------|
| O ₂ v at sub-maximal level of exercise | 180 | 0.600 | .001 |
| O ₂ v at sub-maximal level of exercise excluding all persons with MBC less than 80% of predicted | 88 | 0.771 | .001 |
| O ₂ v at sub-maximal level of exercise holding MBC constant | 180 | 0.757 | .001 |
| MBC holding O ₂ v at sub-maximal level of exercise constant | 180 | 0.849 | .001 |

One can see from Table I that oxygen uptake and ventilation per minute are augmented in a parallel fashion as the intensity of work on the treadmill is increased. Over a very wide range of exercise the subject whose data is presented respired approximately 25 Liters of air for each liter of oxygen uptake as demonstrated in Column 3 of Table I. Oxygen uptake has been clearly demonstrated to parallel physical work except at very high rates of work when anaerobic energy is being expended. One can therefore say that over the low and middle range of work intensity, the ratio $\frac{\text{Minute Ventilation}}{\text{Oxygen Uptake}}$ during steady state of exercise represents the "respiratory response" of the individual to exercise. This ratio is commonly referred to as the "ventilation equivalent" for oxygen and is represented in our data by the symbol O₂v. It is obvious that the smaller this figure is, the

less one will have to use his breathing apparatus for a given stint of physical work. Since this ratio is in essence the "cost" in terms of breathing that one pays for a unit of physical work, one could anticipate that it would have a relationship to maximum ability for oxygen uptake. That this ratio as measured during sub-maximal work correlates rather highly with maximal oxygen uptake is shown in Table VI. If one excludes all persons with a sub-normal maximum breathing capacity (power to breathe), thus choosing a group in whom variation of the O_2v predominates, the correlation is as high as for maximum breathing capacity alone. It is of interest that breathing power as measured by maximum breathing capacity and the cost of physical work in terms of breathing, namely the O_2v , correlate highest with maximum oxygen uptake. The clearcut increase of the correlation when one or the other is held constant by the method of partial correlations is shown in Table VI.

TABLE VII

MAXIMUM O_2 UPTAKE PER SQUARE METER OF BODY SURFACE CORRELATED WITH:

| | <u>No. Studied</u> | <u>"r"</u> | <u>"p"</u> |
|--|--------------------|------------|------------|
| *Maximum Breathing Capacity and O_2v at Sub-maximal Exercise | | | |
| Entire Group | 180 | 0.904 | .001 |
| Normals | 35 | 0.720 | .001 |
| Entire Group Exclusive of Normals | 145 | 0.896 | .001 |
| Emphysema | 28 | 0.892 | .001. |
| Silicosis | 29 | 0.847 | .001 |
| Asbestosis | 19 | 0.849 | .001 |
| Miscellaneous | 30 | 0.940 | .001 |

*Method of multiple correlations

tests which one can use in the field, and they're tackling something which is exceedingly difficult to do, and that is to find a suitable combination of things to define physiological values. They have set about doing something which other people have similarly tried to do, and one of the difficulties has been always in relating a group of tests to define a particular syndrome, if we like, in this case emphysema.

Now, one of the difficulties is that although you may get a high correlation of any one test, unfortunately, the error of prediction is very great. Could I make my point clear with the blackboard? If you have a relationship which they demonstrated, between one function such as, shall we say, a maximum voluntary ventilation and the time of the vital capacitor, then you will find a certain static about the regression line.

Unfortunately, if the correlation is low or even as high as .7, when you predict one from the other, you get a high scatter about this line, and one suggestion that we have tried to work out in overcoming this difficulty, is to use what is, statistically, a discriminate function and that is to combine a series of tests to maximize a difficulty or a difference.

I don't know whether I can make my point clear, but if you will accept for a moment that the clinician can

define gross emphysema as compared with the normal, and Doctor Bristol adequately described that as a possibility, then you can take the different tests, for example, they give maximum ventilation, residual capacity, CO_2 , or the tests predicted from them, and find what combination it might be, three times the predicted M. B. C., plus four times the predicted residual capacity from error of the films, and get a statistical function which maximizes the change from normal to abnormal.

When that is done, you enormously improve the difficulties of any one test being significant, and I would suggest that as a method in using these predicted tests. You can get a discriminate function which gives the maximum difference in accepted normal and extreme emphysema, and then apply that in the field to these other things, using the simple predicted tests. Thank you.

BY DOCTOR McCANN:

Are there any other comments? Doctor Fletcher?

BY DOCTOR FLETCHER:

I just want to ask two simple questions. I was also interested in the age standardization, which, of course, perhaps is not vital, but you did tell us that some men were allowed to stand down from being tested, but you didn't tell us the percentage of the total population that took advantage of that privilege.

The other small point I would just like to correct is your statement that as far as coal miners are concerned, that fibrosis is always associated with emphysema, which is quite a common association, but not always.

BY DOCTOR HUEPER:

What was the labor turn-over in the various groups you tested? I think in dusty occupations, you should usually have your higher turn-over and, therefore, shorter exposure time. If you can manage statistics that way very beautifully.

BY DOCTOR HERMAN:

The general statement was made about dusty occupations. Nothing was said about the degree of dustiness, extreme dustiness, any comments to be made on that?

BY DOCTOR McCANN:

Any other comments? Doctor Bristol, would you like to answer these questions?

BY DOCTOR BRISTOL:

I should like to have Doctor Wright answer these questions, as he is in a better position to do so than I am. Doctor Wright.

BY DOCTOR WRIGHT:

That's all right. The questions have all been, of course, very pertinent. Doctor Bristol took some pains to say this is a preliminary report and when the final

report is made, most of these questions will be answered, in particular the ones about the dust exposure and the labor turn-over.

Neither of those have been the subject of scrutiny as yet. Data is available and is in the process of being looked at. I can say that the - there were a large number of men, I attempted to say the vast majority in the dusty trades, that had been in employment twenty, twenty-five, thirty years. I'm sure that Doctor Vosburgh, who is in the audience, can give you a little more information on that, if anyone wants to ask him personally. That will be in the final report.

The age distribution, I can tell you roughly about. It ran from approximately twenty-five to seventy and we took pains in choosing our control group, to have the same age distribution.

Now, the matter of obtaining volunteers in the tool and dye and punch press, in the dusted trades, we obtained all of the people except two in the foundry, and eight in the porcelain. Those were people who were away on holidays and we couldn't get them. There were no refusals, and I don't think that our relation was seriously impaired by the lack of those ten individuals. There were relatively few individuals who refused when requested, in the tool and dye and punch press. That was a random sample and then the

people picked were asked if they would volunteer. There were some refusals; I can't tell you precisely how many.

I'm afraid that maybe we won't be able to tell you that at all, because I'm not sure that records were kept as to how many refused, but I will look into that. It's a good question and, for the sake of completeness, we should have it.

Doctor Hugh-Jones' suggestion is a good one. You will see in the next paper that we attempted to get at that to some extent by full time correlation procedure. I'm not certain that that's the same thing he's talking about. I believe that his procedure is a little bit different and it is one that I'm not familiar with. I certainly will get together with you afterwards and see if we can't improve our data.

Did I leave anybody out?

BY DOCTOR BRISTOL:

The complication of conglomerate.

BY DOCTOR WRIGHT:

Oh, yes, perhaps Doctor Bristol didn't make it clear enough. I think he did say, commonly or almost always, in regard to emphysema being a complication of conglomerate disease. We certainly have had the experience in the vast majority of conglomerate silicotics, of demonstrable evidences of emphysema. We have one, I published a

very striking case of no emphysema.

BY DOCTOR FRIEDMAN:

Doctor Wright, in the dusty trade groups that you analyzed, what percentage of significant pulmonary dust disease did you find in the dusty groups?

BY DOCTOR WRIGHT:

You mean how many have radiological evidences of silicosis?

BY DOCTOR FRIEDMAN:

How many have radiologic evidences and how many were disabled by our other standards of function studies?

BY DOCTOR WRIGHT:

Well, insofar as the number who had radiologic evidences, there were some. That will be in the final report. Doctor Bristol is just getting ready now to review on large film all of these cases. There were some, perhaps Doctor Vosburgh could give us a rough estimate. Other methods of study were not carried out in these individuals. I wish that such an opportunity were made available to us.

BY DOCTOR FRIEDMAN:

Now, in the dusty trades, you analyzed then, there isn't necessarily a very great hazard that could have been corrected previously with proper hygienic industrial methods. You may have entered into places where improvement had already taken place.

BY DOCTOR WRIGHT:

On all of these people, I think the employment records will show they have been there twenty, twenty-five years.

BY DOCTOR FRIEDMAN:

And you know how many years they worked in hazard-our concentration and how many years not?

BY DOCTOR WRIGHT:

Yes, we have a letter - we have all that information on cards, and it is being gone over now. That will be in the final report.

BY DOCTOR McCANN:

If there is no further discussion, we will proceed to the next paper, and we're going to change the order of the program so that Doctor Wright's paper will precede that of Doctor Gregoire. Doctor Wright.

BY DOCTOR WRIGHT:

Mr. Chairman, Ladies and Gentlemen: Before presenting the material in my paper, I would like to make one comment to amplify, to some extent, one of the things that I think was rather important that was brought out by our Chairman, and that is the importance of the clinician in these various studies, and the position that gadgeteering and attempts to measure with some precision has in such studies as we are making.

INTER-OFFICE COMMUNICATION

DATE: January 23, 1953

TO: George W. Wright, M.D.

FROM: Arthur W. Vorwald, M.D.

SUBJECT:

Dear George:

Attached is the stenotypist's copy of your paper for the Seventh Saranac Symposium. Please return it with your formal paper just as soon as you possibly can, together with Gregoire's material.

January 20, 1953

MEMORANDUM:

TO: DR. GEORGE W. WRIGHT

FROM: DR. A. J. VORWALD

Dear George:

This is to remind you again that we have not as yet received your paper for the Seventh Saranac Symposium, as well as Fernand Gregoire's material, which he has informed us he sent to you on December 13. Not having these papers is seriously handicapping the publication of this conference. Please forward your material and Gregoire's as soon as possible.



AJV:gn

Art!

*Am working on Gregoire paper.
I will not be able to do anything on
mine until I receive the stereotype
copy from you!*

Jean

January 20, 1953

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TO: DR. GEORGE W. WRIGHT

FROM: DR. A. J. VORWALD

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Paper by Wright

sent, but not in

Chapter Twenty-four

Maximum Ability for Physical Work of Moderate Duration Correlated with
Various Tests of Pulmonary Function

George W. Wright, M.D.

Before presenting the material.....

.....

.....taken a part in it.

sent
but
not
in

Feb 3 '55
Original sent
to DR Wright

MAXIMUM ABILITY FOR
OF MODERATE DURATION COR
TESTS OF PULMON

George

Dr. Wright's talk on
Compensation for
Disability

pages 553 to 564 inclusive
(He must have them)

Dr. Wright
Do you wish to answer
Dr. Herman's question about the
degree of dustiness (p. 501)?
If so, add question and answer
on p. 90

* Head, Department of Physiology, The Edward L. Trudeau Founda-
tion, Saranac Lake, New York

Feb 3.55
Original sent
to DR Wright

MAXIMUM ABILITY FOR PHYSICAL WORK
OF MODERATE DURATION CORRELATED WITH VARIOUS
TESTS OF PULMONARY FUNCTION

George W. Wright*

* Head, Department of Physiology, The Edward L. Trudeau Foundation, Saranac Lake, New York

Mr. Chairman, Ladies and Gentlemen: Before presenting the material in my paper, I would like to make a comment to amplify one of the things brought out by our Chairman that I think was rather important.

I find myself in full agreement with him as regards the importance of the clinician and the questionable tendency we all have to rely too strongly on laboratory procedures. This reliance in clinical medicine is caused in part by the large number of patients that the physician must see daily. I think it is also to some extent due to the fact that we can see and understand figures more easily than we can those clinical observations that go into forming an opinion. I do believe, however, that Doctor McCann would be the first to say that we need objective laboratory methods of measurement if for no other reason than to apply some sort of a test to our clinical abilities.

The clinician has for years expressed "opinions" but has lacked more precise methods of confirming his opinion. One of the most important uses of more precise methods of measurement is to apply them as a test of our clinical abilities. It is partly in this vein that our work has been carried out and the study that I am to report this morning was done.

May I have the first slide? (Fig. 1) Because this is a mixed audience, I am going to take just a few moments to prepare some background for the comments that I am going to make and I have hopes that my medical colleagues will forgive me for this crude diagram which, to my mind, portrays in an easily understood

manner the vital functions of respiration.

The respiratory apparatus is, in essence, a bellows or air pump through which blood circulates for the purpose of being ventilated. Air enters into the bellows and is then distributed throughout the bellows. Being constantly replaced by each breath, the lung air is maintained with a higher pressure of oxygen and a lower pressure of carbon dioxide than exists in the blood that flows through it. The venous blood returns to the lungs from the periphery where it has given up part of its oxygen and taken on additional carbon dioxide. As the blood circulates through the bellows, gas passes across a very thin membrane which separates the blood phase from the gas phase of the lung, oxygen going into the blood and carbon dioxide coming out. The blood then returns to the left side of the heart and is pumped out to the tissues, thus completing the circuit for oxygen and carbon dioxide transport. In essence, the respiratory apparatus is an air pump and a diffusion surface. It is intimately associated with and cannot be separated from the circulatory system which pumps the blood into the lung, accepts blood back from the lung and then pumps it out to the tissues.

The vulnerable points of such an apparatus are immediately apparent. The bellows itself is vulnerable. Loss of power to actuate it or loss of deformability or the ease with which its volume can be changed will obviously impair its utility as a pump. The apparatus is also vulnerable in regard to the movement of air through the airways. Any narrowing of these will obviously interfere with the capacity of this apparatus to behave

as an air pump.

Improper distribution of air in this bellows will lead to regions poorly ventilated and possibly even areas of lung that are not ventilated at all. If these poorly ventilated areas are still circulated, the venous blood that flows through them does so to no purpose whatsoever. The system is vulnerable at the thin membrane which separates the blood from the gas phase. If this becomes thickened or otherwise changed, you can readily understand that gases may have trouble in getting across this barrier. The circulatory system itself is vulnerable. The total area which can receive blood or the cross-sectional area of the vascular bed may be so diminished that blood is pumped through with considerable difficulty. Moreover, blood may dam back in the lung because of failure of the pump on the left side to move the blood along as it leaves the lung. The circulatory aspect is going to be covered by Doctor McCann.

During the past twenty years, methods have been developed which will permit a fairly precise measurement of the activities of the respiratory apparatus, especially the study of those vulnerable points that I have just mentioned. I would like to say that Doctor McCann and his group were and continue to be pioneers in this type of study.

Some thirteen years ago, Doctor Gardner became interested in the effects that the inhalation of dust might have on the function of the lung and circulation. It was because of this interest that our physiology laboratory was developed. We began our studies by measuring the individual actions of the respiratory

apparatus. These procedures are those that you already have been told about by others that have talked on the subject of pulmonary function during this symposium.

We measured the size of the bellows at various degrees of enlargement, the so-called pulmonary volumina; we measured the stroke volume of the air pump, the vital capacity; we measured the power that this pump has to deliver air per minute, namely, the maximum breathing capacity. We have attempted, with relatively little success, to measure the distribution and mixing of gases in the lung. With some greater success, we have been able to measure the impairment, if any, for the passage of gases from the gas phase to the blood phase. This is done in two ways, first, by studying the degree to which the hemoglobin is saturated with oxygen and the degree to which carbon dioxide has been removed from the blood after it has passed through the lungs and, second, we have measured the pressure head that exists between the oxygen and carbon dioxide in the gas phase and the oxygen and carbon dioxide in the blood phase.

Unfortunately, we have not been able to measure with nearly the same degree of accuracy the role of the circulatory apparatus. We will be much further ahead when maximum cardiac outputs can be measured and when the pressure relationships about which Doctor McCann will tell you, that govern the flow of blood through the lung, can be measured under conditions of severe stress.

After about four years of work, measuring these individual functions of the lung, we came to the realization that we were

not really accomplishing all that we had set out to do. It is true that we could recognize failure of the blood to be properly ventilated. We could recognize impairment of the pumping action of the respiratory apparatus. We could recognize poor distribution of gases. We and others have referred to these specific abnormalities of function by using the general term of "disability" without making it absolutely clear that the disability was for a specific function. A low maximum breathing capacity is a disability for breathing power. A subnormal arterial saturation is a disability for blood gas transfer. We have often confused the issue by failing to say whether or not this specific disability as regards breathing power or ventilation of the blood is actually a disability in terms of a man's ability to work. During about four years of effort along these lines, we were repeatedly struck with the fact that certain men were remaining on the job doing a full day's work in spite of considerable impairment of breathing power, some degree of arterial unsaturation and abnormalities of the pulmonary volume. It took us a long time to realize that we should be much more careful in the use of the word disability and should define it precisely every time that it is used.

The word itself means exactly what it says, loss of an ability, and we should be careful to say what ability. We, as have many others before us, cast about for some way of trying to relate these specific disabilities to the general ability of a person to perform as a physical engine.

There are various measurable things that reflect a person's ability to "work." The earning power of a man is to some extent

a measure of his ability to perform as a physical engine but it is not a very precise one.

As doctors, we are of course interested in the human body and therefore desire to find a more direct and precise way of measuring what a man can do in the way of maximum physical performance. Some work has been done along the lines of measuring peak abilities for physical output. For example, the records that have been set in distance running and in other forms of physical exercise are, in a sense, a measure of this specific ability.

There is a more practical and generally applicable way of getting at one's maximum capacity for physical work. The human engine is a great deal like an ordinary internal combustion engine. Fuel is burned, oxygen is required in that burning, and carbon dioxide is the result of that burning. Just as you might rate the energy spent by any engine in terms of its oxygen consumption, the human body can be studied in essentially the same way.

Figure 2 shows the manner in which one can study maximum performance abilities by having the individual walk on a treadmill rather than run on a circular track. Much excellent work has been done in this manner at the Fatigue Laboratory at Harvard studying athletes and some of that data I will show you in just a moment.

We have studied normal and diseased men with respect to their greatest ability to put out physical energy by having them walk on a treadmill. We chose that form of stress rather than

the bicycle because in this country our people are not accustomed to riding a bicycle.

Figure 2 shows a man walking on a treadmill while various measurements are being made. Studying the individual in this way, at increasing intensities of physical exertion, certain measurements can be made as shown in Table I. This is the data of a normal person who has been walked at a constant speed at three and a half miles an hour but increasing the grade on which he walked until finally a grade is reached which is the utmost that he can accomplish. In other words, at the next higher grade the walk could not be completed because of intolerable distress.

These walks were all of six minutes' duration and by increasing the grade on which the man walked we finally reached a point where the man could not walk for the full six minutes. Normal individuals terminate the walk because of severe leg weakness and breathlessness.

It is apparent that the human engine is quite similar to the internal combustion engine. The oxygen uptake (Table I), as you would expect, increases and gets up to a peak oxygen uptake of 2.950 cc. of oxygen per minute. This is the greatest ability that this individual had for oxygen uptake. There is very little increment in the last two intensities of exercise. Our data show that a plateau is reached in oxygen uptake at a time when the ventilation of the lungs can still be augmented. This, with other evidence, has led to the conclusion that in the normal man the circulation is the governing factor for peak physical output of moderate duration. We're not talking about short bursts of energy output.

It is generally accepted that one of the measures, and perhaps the best at present available, of a man's ability to perform as an integrated being is the height to which he can increase or raise his oxygen uptake during exercise. This is certainly a function not only of respiration but also of circulation and I believe in normal persons is primarily a function of the circulation. The other tables that I will show you will indicate, as Doctor Motley stated, that we have enormous respiratory reserves; they are far greater than those for circulation.

We have chosen the peak oxygen uptake as our measure of the maximum ability of the human engine to perform. Each man studied in the data that I will show you was carried in a series of walks to his peak oxygen uptake. The first two or three exercises can be done in one day but subsequent, more severe ones should be done one or at most two a day.

In Figure 3 are shown the maximum oxygen uptakes of 47 normal men. The oxygen uptake in liters per square meter of body surface is plotted on the ordinate and the age in years on the abscissa. There is a loss of this ability as the man becomes older, a fact of common experience. The degree of spread is interesting in that I had anticipated there would be a greater variation. The standard deviation is approximately 13% of the mean.

Figure 4 shows some data from abnormal individuals in respect to peak oxygen uptake plotted on the "grid" of normal persons. The heavy line defines the expected normal maximum oxygen uptake based on age in years. The grid embraces two

standard deviations on either side of the mean. The resting oxygen uptake and the amount of oxygen intake for a level walk are indicated in order to show the reserves we possess. To support the belief that peak oxygen uptake is a good measure of a man's ability to perform physical work, I have plotted data taken from a paper that appeared in Science by Robinson, Dill and their co-workers on peak power of physical performance of some of our fine athletes.

The open circle at the very top on the far left is the data from Don Lash, who many of you will remember was our very fine two-mile and cross-country runner. The three that are plotted below him are athletes who were also very fine distance runners. It is apparent that these men have a phenomenal ability for oxygen uptake. This is an ability that is far beyond anything that is possessed by the rest of us mere mortals, the average normal individual's ability being indicated by the heavy regression line. The remainder of the data is of some pathological subjects. These should not be considered as necessarily representing a true cross section of each of the diseases represented. While the group as a whole shows less ability than that possessed by normal males, many still fall within normal limits and nearly all still possess more than enough ability to walk on the level at 3.5 mph. It is of interest note that the young man with only one lung (a pneumonectomy and ~~thoracoplasty~~ ^{thoracoplasty} having been performed for tuberculosis) still possesses such a large ability for maximum physical energy output.

To what extent do the various static and dynamic characteristics of the respiratory apparatus influence or correlate with a person's maximum ability for physical energy output? During the past few years we have measured the maximum oxygen uptake ability using the method we have just described in 180 individuals. These same persons also were subjected to various "tests" of pulmonary function. The group includes normal persons and those having various diseases of the respiratory apparatus. There were no unusually skilled athletes or "trained" persons in the group. All degrees of respiratory and physically impaired persons were included except those so impaired that they could not even walk at 1.25 mph. on the level for six minutes. We deliberately excluded all persons with overt cardio-circulatory disease. It is likely that a few persons with unrecognized mild cor pulmonale or with disease of the coronary vessels may have been included because of the age groups and the nature of the pulmonary diseases studied.

TABLE II

MAXIMUM O₂ UPTAKE PER SQUARE METER OF BODY SURFACE CORRELATED WITH:

| | <u>No. Studied</u> | <u>"r"</u> | <u>"p"</u> |
|-----------------------------------|--------------------|------------|------------|
| Vital Capacity, Absolute Value | | | |
| Entire Group | 176 | 0.691 | .001 |
| Normals | 38 | 0.486 | .001 |
| P ₁ and P ₂ | 21 | 0.511 | .02 |
| Silicosis | 29 | 0.533 | .01 |
| Asbestosis | 18 | 0.495 | .05 |

TABLE II (continued)

| | | | |
|---|-----|-------|------|
| Miscellaneous | 26 | 0.836 | .001 |
| Emphysema | 25 | 0.273 | .100 |
| "Be" Granuloma | 10 | 0.468 | .100 |
| Vital Capacity as % of Predicted Normal | | | |
| Entire Group | 176 | 0.696 | .001 |
| Normals | 38 | 0.159 | .100 |
| P ₁ and P ₂ | 21 | 0.610 | .01 |
| Silicosis | 29 | 0.481 | .01 |
| Asbestosis | 18 | 0.619 | .01 |
| Miscellaneous | 26 | 0.721 | .001 |
| Emphysema | 25 | 0.546 | .01 |
| "Be" Granuloma | 10 | 0.433 | .1 |

Tables II, III, IV, V, VI, and VII show the correlation coefficient "r", and level of significance "P" for the relationship of several of the commonly performed "tests" of the respiratory apparatus to maximum oxygen uptake.

Table II shows the degree of correlation between Vital Capacity, both absolute and as a percent of predicted normal value, to be moderately good for the group as a whole. When broken down into sub-groups on the basis of diseases, a significant correlation does not always exist. The Vital Capacity has been used for years as a method of estimating "disability" but its failure to correlate with "clinical experience" in many instances has come to be recognized. The data in Table II substantiates this experience.

TABLE III

MAXIMUM O₂ UPTAKE PER SQUARE METER OF BODY SURFACE CORRELATED WITH:

| | <u>No. Studied</u> | <u>"r"</u> | <u>"p"</u> |
|--|--------------------|------------|------------|
| Residual Capacity as % of Total Capacity | | | |
| Entire Group | 174 | -0.551 | .001 |
| Normals | 41 | -0.353 | .05 |
| Emphysema | 25 | -0.688 | .001 |
| Silicosis | 29 | -0.693 | .001 |
| Asbestosis | 17 | -0.119 | .1 |

Impediment to air flow through the tracheo-bronchial tree, especially during the expiratory phase, reduces the maximum breathing capacity and in the majority of instances diminishes the degree to which the lungs can be emptied. Abnormal volumes of air retained in the lungs at the end of the most powerful expiration possible can be detected by expressing the residual capacity as a percent of the total capacity of the lungs, except in those few examples in which the ratio is abnormally high by reason of a pathologically small total capacity. The ratio of R.C./T.C. x 100 correlated with maximum oxygen uptake is shown in Table III. It is well established that a high R.C./T.C. ratio is a consistent finding in diffuse obstructive emphysema; hence it is not surprising that a highly significant correlation is found for the entire group because it contained a large number of emphysematous persons. The significant correlation in the emphysema group per se suggests strongly that the degree of abnormality of the R.C./T.C. ratio parallels the loss

of ability for maximum oxygen uptake. The high correlation in the silicotic group is undoubtedly due to the fact that diffuse obstructive emphysema existed in a number of this group. The lack of correlation in the normal and asbestosis groups fits well with the fact that diffuse obstructive emphysema of a clinically significant grade is absent in these persons.

TABLE IV

MAXIMUM O₂ UPTAKE PER SQUARE METER OF BODY SURFACE CORRELATED WITH:

| | <u>No. Studied</u> | <u>"r"</u> | <u>"p"</u> |
|---|--------------------|------------|------------|
| Maximum Breathing Capacity (Absolute) | | | |
| Entire Group | 180 | 0.755 | .001 |
| Normals | 41 | 0.508 | .001 |
| Entire Group excluding Persons with MBC over 150 L/min. | 150 | 0.696 | .001 |
| Emphysema | 26 | 0.797 | .001 |
| Silicosis | 29 | 0.802 | .001 |
| Asbestosis | 17 | 0.590 | .02 |
| Maximum Breathing Capacity as % of Predicted Normal Value | | | |
| Entire Group | 180 | 0.559 | .001 |

The maximum breathing capacity measures the maximum ability of the respiratory apparatus to behave as an air pump. Its correlation with maximum oxygen uptake is shown in Table IV. For the group as a whole, "r" is higher than for vital capacity or for the ratio of R.C./T.C.. As might be anticipated, the group of normal persons did not show as high a correlation as

did those groups containing a high proportion of emphysematous persons. This finding supports the concept that in normal persons the respiratory power is in excess of the circulatory and hence only when disease diminishes the respiratory power does the maximum breathing capacity become a strongly significant determinant of maximum oxygen uptake. The high correlation between maximum breathing capacity and maximum oxygen uptake in emphysema either per se or as a complication of silicosis is to be expected since in this pathological condition severe airway obstruction and limitation of breathing power exists as the primary cause of exertional dyspnea which in turn limits the patient's ability for physical work. The low significance of the correlation in the asbestosis group reflects the minor role that diffuse obstructive emphysema plays in this disease.

As shown in Table IV, no advantage is gained by expressing the maximum breathing capacity as a percent of the normal value predicted for each person. In fact, the coefficient is somewhat lower.

TABLE V

MAXIMUM O₂ UPTAKE PER SQUARE METER OF BODY SURFACE CORRELATED WITH:

| | <u>No. Studied</u> | <u>"r"</u> | <u>"p"</u> |
|--|--------------------|------------|------------|
| Arterial Hemoglobin Saturation during Exercise | 156 | 0.446 | .001 |
| Arterial-Alveolar pO ₂ Difference during Exercise | 148 | -0.372 | .001 |
| Drop in Arterial CO ₂ Content during Exercise | 92 | 0.498 | .001 |

TABLE I

METABOLIC AND RESPIRATORY RESPONSE TO WORK OF INCREASING INTENSITY*

| Consumption c./minute | Ventilation liters/minute | Ventilation Equivalent (O ₂ v) | Dyspnea Index ($\frac{M.V.}{M.B.C.} \times 100$) | Exertional Dyspnea (Subjective) | Work Intensity m.p.h./grade |
|--------------------------|------------------------------|--|---|------------------------------------|--------------------------------|
| 1290 | 29.8 | 23.1 | 16 | 0 | 3.5/level |
| 1570 | 36.0 | 23.0 | 19 | 0 | 3.5/2% |
| 1620 | 40.6 | 25.0 | 22 | 0 | 3.5/4% |
| 1820 | 45.2 | 24.8 | 24 | 0 | 3.5/5% |
| 1995 | 48.5 | 24.3 | 25 | 0 | 3.5/6% |
| 2200 | 52.2 | 23.7 | 28 | 0 | 3.5/7.5% |
| 2300 | 59.8 | 26.0 | 32 | 1+ | 3.5/8.5% |
| 2715 | 71.0 | 26.1 | 37 | 3+ | 3.5/10% |
| 2900 | 88.2 | 30.4 | 47 | 4+ | 3.5/12% |
| 2950 | 96.1 | 33.2 | 52 | 4+ | 3.5/14% |

* Normal man, age 39, weight 178 pounds, height 71 inches. Work performed on motor driven treadmill, expired air being collected in the 5th and 6th minutes of exercise.

The effectiveness with which the respiratory apparatus ventilates the blood is reflected by the degree to which the hemoglobin of arterial blood is saturated with oxygen, the head of pressure for oxygen across the membrane separating the blood from the gas phase of the lung as expressed by the arterial-alveolar pO_2 difference during exercise and by the difference in arterial carbon dioxide content at rest and during exercise. The correlations of these factors with maximum oxygen uptake are shown in Table V. Although there is a significant correlation in all three, it is generally of a low order. It is probable that when this data is further examined by breaking the group into sub-groups according to diseases, there will be in some no correlation at all and in others a better correlation. The concept that the arterial-alveolar pO_2 difference and the degree to which arterial hemoglobin is saturated with oxygen constitute good measures of a person's ability to do physical exercise is not substantiated by our data. (carried out in our laboratory and) In fact, as shown in the asbestosis study reported herein by Doctor Gregoire, arterial hemoglobin unsaturation does not presage a lowered ability for maximum oxygen uptake. This will not seem strange when one recalls that great civilizations have been developed at altitudes so high as to make one certain that all of the inhabitants had arterial hemoglobin unsaturation. The pathological respiratory conditions, for example low maximum breathing capacity, that lead to or accompany the unsaturation of hemoglobin are likely stronger determinants of maximum ability to use oxygen than is hemoglobin unsaturation per se.

Since maximum breathing capacity and O_2v are ~~linearly~~ ^(highly correlated) and entirely independent physiologic factors, we have, by multiple correlation technique, studied their combined relationship to maximal oxygen uptake as shown in Table VII. The very high correlation for the entire group is maintained when specific sub-groups are studied.

TABLE VIII

REGRESSION EQUATIONS FOR PREDICTING MAXIMUM O_2 UPTAKE FROM:

| | |
|-----------------------------|--|
| Vital Capacity: | $O_2/m^2/min. = 324 + 234 \text{ V.C.}$ |
| | S.D. = 236, Coeff. of variation = 21% |
| Maximum Breathing Capacity: | $O_2/m^2/min. = 424 + 6.26 \text{ M.B.C.}$ |
| | S. D. = 220, Coeff. of variation = 19.8% |
| M.B.C. + O_2v : | $O_2/m^2/min. = 1159 + (5.75 \text{ M.B.C.} - 21.8 \text{ } O_2v)$ |
| | S.D. = 143, Coeff. of variation = 13% |

The scattergram of the correlations between Vital Capacity, Maximum Breathing Capacity, and M.B.C. + O_2v with maximum oxygen uptake are shown in Figs. 5, 6 and 7 respectively. It is apparent that although the correlation coefficient is significantly high in all, the scatter is much greater for Vital Capacity than for Maximum Breathing Capacity or M.B.C. + O_2v and is least for the latter. The regression equations for these three correlations are shown in Table VIII.

These data strongly suggest that M.B.C. + O_2v are the principal physiologic determinants of maximum capacity for oxygen uptake in persons having pulmonary disease without overt circulatory disease.

as a complication. Exercise to a maximum tolerated level is time consuming and not always practical as a means of measuring the overall ability of the human engine to expend energy. The importance of the M.B.C. + O_2v as measurable characteristics from which one can properly estimate the normality of a person's ability for physical energy output is apparent.

These data also show that whereas abnormality of the vital capacity, arterial blood gases, etc., and even abnormality of the maximum breathing capacity or O_2v per se may be "disabilities" for the specific function measured, one must question the correctness of presuming that such abnormalities of necessity indicate a disability for physical energy output. When the departure from normal for any of these specific characteristics of the respiratory apparatus is gross, the correlations indicate a high probability that the maximum oxygen uptake will ^(also) be sub-normal. When, however, the departure from normal is small, the maximum oxygen uptake may still be well within the normal range.

The fact that the circulatory system has not been critically assessed in these studies must be borne in mind, especially if practical applications of the information are to be made.

In closing, I would like to state that this work is a preliminary report and was contributed to by the entire laboratory personnel over the last few years. It has been my privilege to report it.

Legends for Dr. George Wright's Paper

Maximum Ability for Physical Work of Moderate Duration Correlated With Various Tests of Pulmonary Function

- Fig. 1. Schematic diagram of essential mechanical factors of respiration.
- Fig. 2. Physiologic data being obtained while subject exercising on motor-driven treadmill.
- Fig. 3. Maximum oxygen uptake of normal men plotted against age in years.
- Fig. 4. Maximum oxygen uptake of abnormal individuals plotted on "grid" of normal persons.
- Fig. 5. Scattergram of maximum oxygen uptake plotted against vital capacity in a group of normal and pathological subjects.
- Fig. 6. Scattergram of maximum oxygen uptake plotted against maximum breathing capacity in a group of normal and pathological subjects.
- Fig. 7. Scattergram of maximum oxygen uptake plotted against a combination of maximum breathing capacity and oxygen ventilation equivalent.

Georg Wengert's
Paper

7. Fig

" Maximum Ability For
Physical Work

→

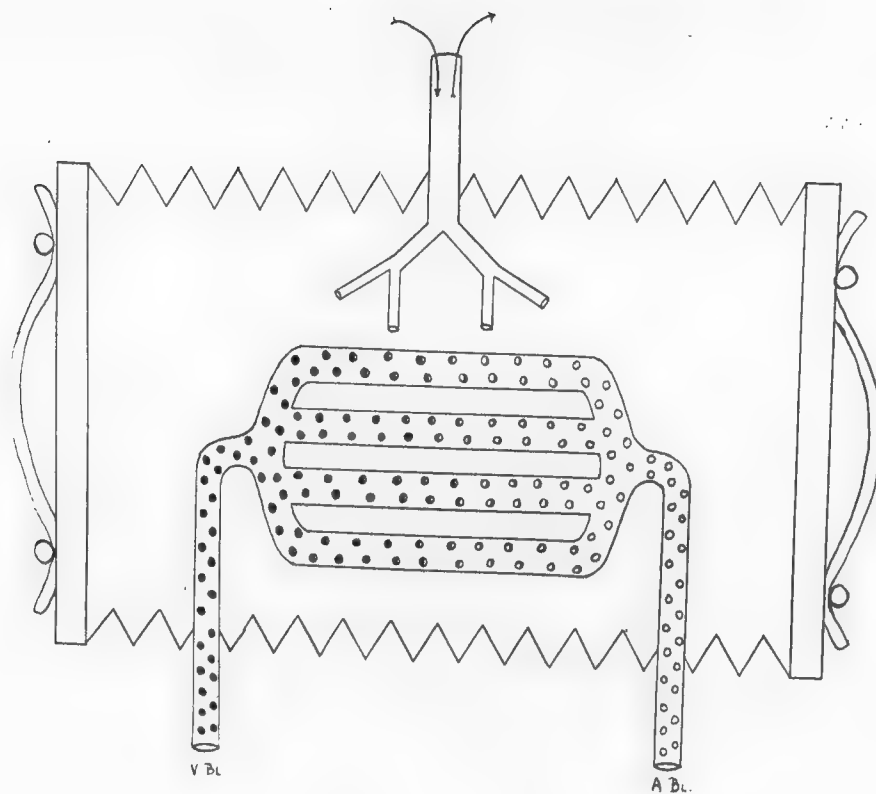


Figure 1



Figure 2

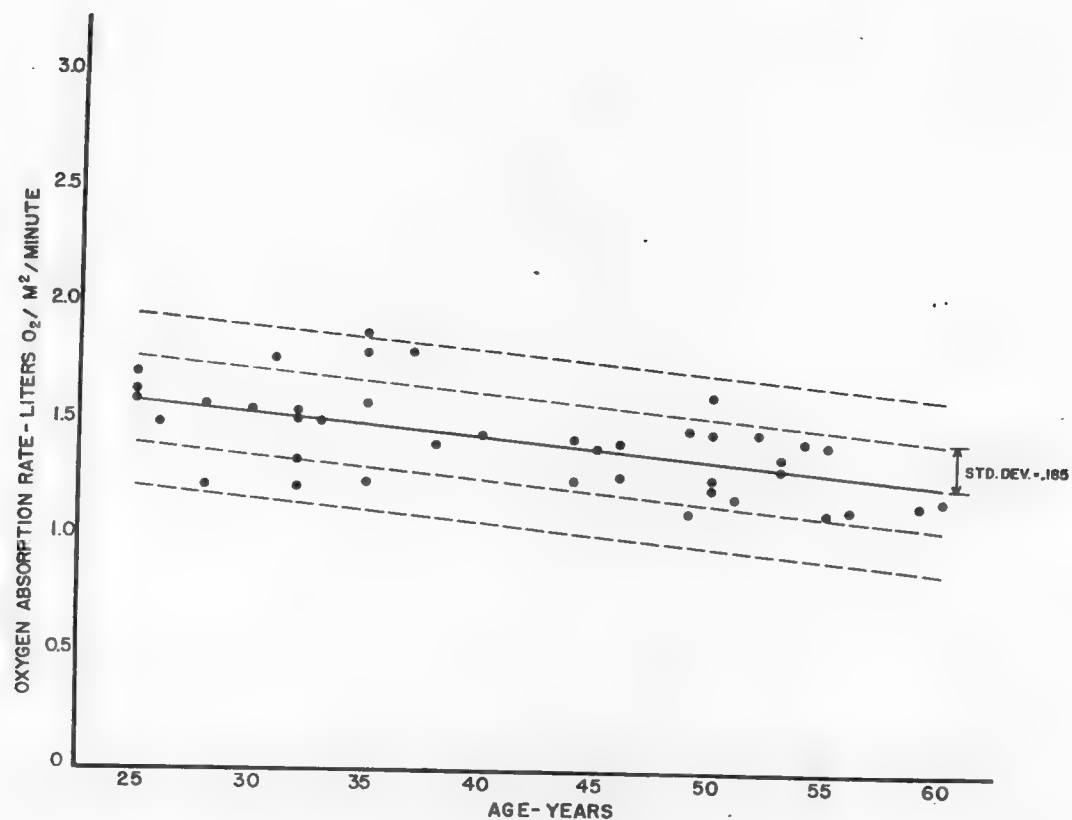


Figure 3

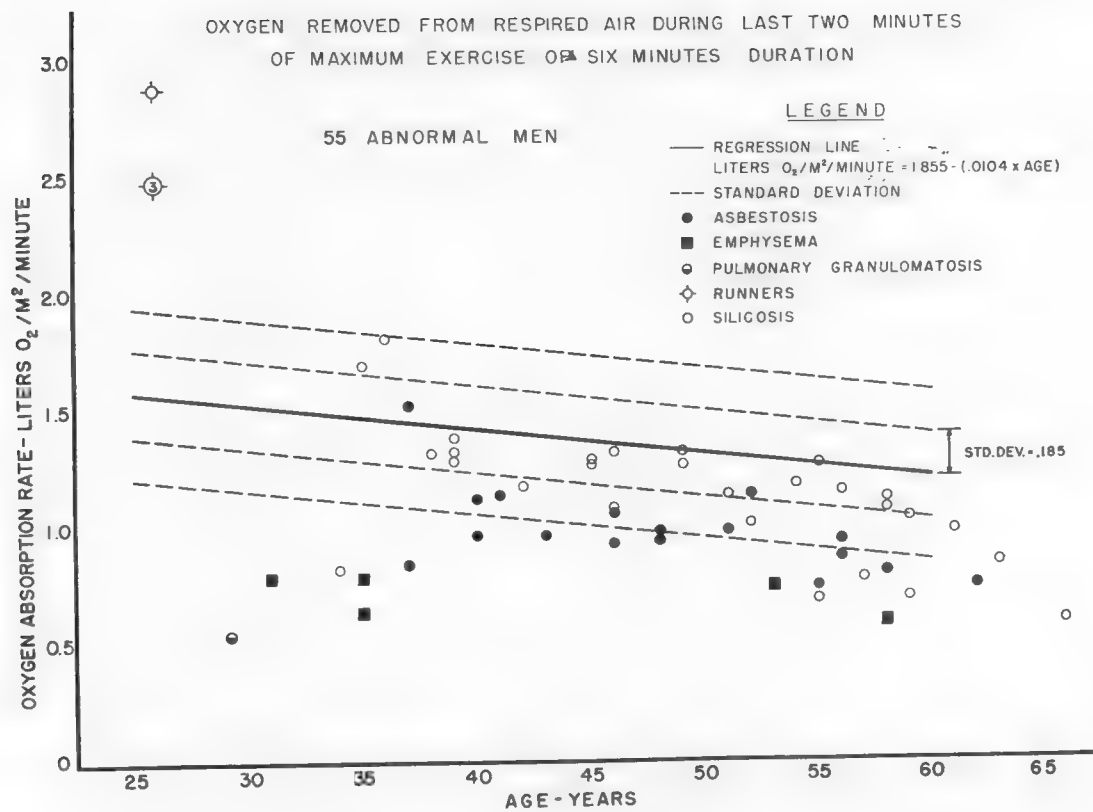


Figure 4

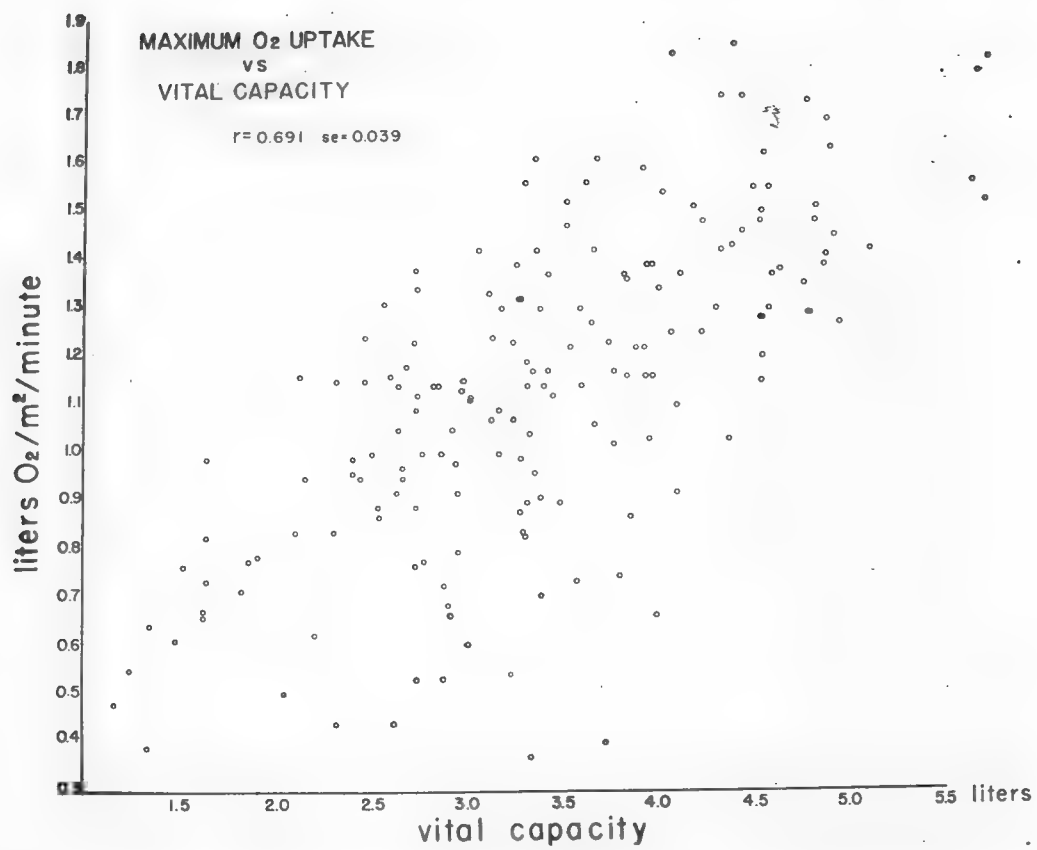


Figure 5

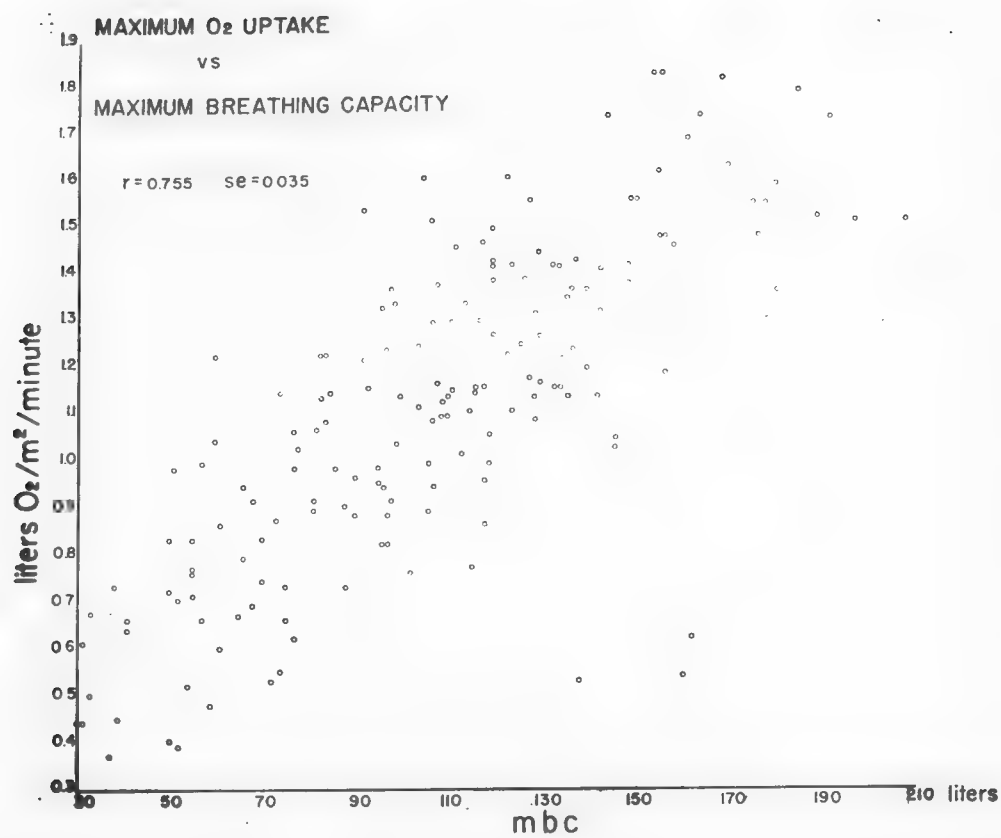


Figure 6

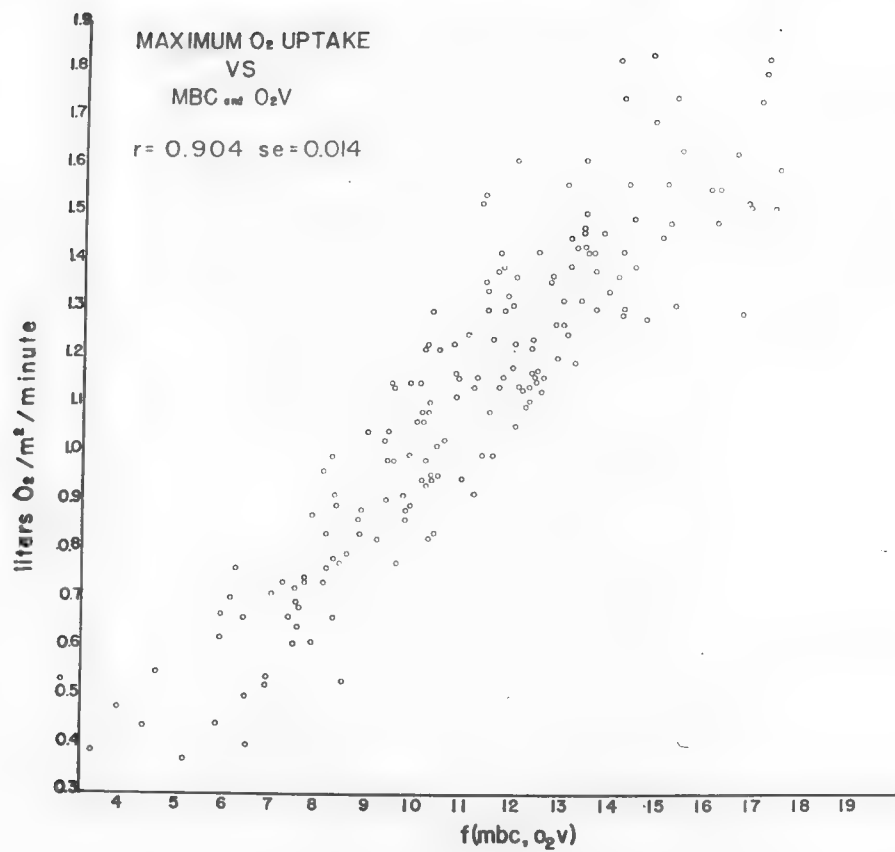


Figure 7

Chapter Twenty - four

Maximum Ability for Physical Work of Moderate Duration Correlated with
Curious Tests of Pulmonary Function

George W. Wright, M.D.

Before presenting the material.....

.....taken a part in it.

Pulmonary Function Studies in Men Exposed for Ten or More Years to
Inhalation of Asbestos Fibers

Fernand Gregoire, M.D.

(Neither transcript nor paper on file)

Discussion

*But references
at bottom
of page indicate*

DR. HUGH-JONES: Dr. Wright offered a suggested "definition", shall we say, of measuring a man's overall capacity for work. Taylor (1931) and Briggs (1919) measured what they called the "crest load", which is the maximum work that a man can do in a steady state, and others have found the maximum ability to work as measured by the oxygen consumption, like Dr. Wright did. But one thing worries me: Although two persons may have very different powers to do their maximum effort, does that fact necessarily mean they are limited in the same way at the ordinary work they do every day? Does Dr. Wright have evidence to show that the performance at sub-maximal loads is necessarily worse for people who show a falling-off of their maximum ability? It doesn't seem to me that this must necessarily follow.

If you will forgive me, as a mere physiologist, I should also like to suggest two technical criticisms of the statistical methods. The first is in the use of the correlation coefficient. Dr. Wright was surprised at the extremely high coefficient he got between the maximum breathing capacity, combined with the ventilation equivalent for oxygen, and the maximum power to work as measured by

oxygen consumption. I believe that this may possibly be a "spurious correlation". He has the same factor entering the top and the bottom of a fraction, namely, the ventilation in maximum breathing capacity in one case and the ventilation equivalent in the other. Further, I believe that in his examples of comparing correlations he should have used partial correlations throughout. You can use the overall correlation as a measure of the amount one test is related to some other second test. But, if you are comparing a third test to the first - in this case the oxygen capacity for work - the reason for a high correlation may be because the third is highly correlated with the second test. I do not believe you can make real comparisons on that basis unless you use partial correlations and particularly be wary of any spurious correlations which may arise from the use of fractional indices.

The second comment is regarding Dr. Gregoire's statement that these tests are "significantly different". I should like to ask precisely what he means. If he used twice the standard error of the difference as a significance test (I believe he did) he assumed a normal distribution curve for his results. Yet he was using for some comparisons a fraction which I cannot believe was normally distributed if its components were. I wish to ask also what he takes as a level of statistical significance when the distribution curve is skewed.

Both, Dr. Wright and Dr. Gregoire, in presenting their data, showed a regression line with the test results scattered about it. I entirely agree with this presentation. It is much better, it seems to me, than the practice of expressing things as a "percentage of the predicted normal". Instead of saying that somebody has a test result which is "100 per cent of a predicted normal", we should say that the result lies within plus or minus a certain scatter of what would be expected for his age on the average. In fact in correcting for an age effect, one should subtract the average fall with age and not express the change as a percentage. * Tanner (1949) criticizes physiologists for this very common error. If we are going to use statistical methods, I think we should consult statisticians in order to avoid drawing erroneous conclusions.

* Tanner J.M. (1949) J clin Invest. 28 567. 7

I was extremely interested in Dr. Gregoire's conclusions about the differences between silicosis and asbestosis and I have no doubt at all about their validity; I hope my comments about the individual significance tests do not suggest that I had.

DR. WRIGHT: Dr. Hugh-Jones, I'm afraid you read more into my paper than I intended to have in it. You have gone beyond the limits of the paper and have applied the peak oxygen uptake to a man's ability to shovel coal. I refrained deliberately from doing that because I wished to discuss only what we measured. The factors related to a man's ability to perform his everyday tasks are most complex - his motivation, how many children he must feed - and why people become fatigued on their job is a most perplexing question. Why, in this era of labor-saving devices, is the housewife who may not have done much physical work

See notes at Beginning of the discussion

example, I might have a reduced maximum ventilation or maximum ventilatory power of my lungs, and, therefore, I might not be able to run a mile as fast as I formerly could, but I might be able to walk at a ^{slow} rate for a long time.

I should like to make two technical criticisms of statistics. The first is on the use of the correlation coefficient. Dr. Wright was surprised at the extremely high coefficient he got between the maximum breathing capacity, combined with the ventilation equivalent for oxygen, and the maximum power to work. I believe that is an example of what is technically called a spurious correlation. He has the same factor entering the top and the bottom of the fraction, namely, the ventilation in maximum breathing capacity in one case and the ventilation equivalent in the other. I believe that in his examples of comparing correlations he should have used partial correlations throughout. If you compare the overall correlation, what you are observing is the amount one test relates to some other test, and the degree of that comparison is a set thing when you are comparing just one set of tests. If you are comparing another test to the first test - in this case the oxygen capacity for work - the reason for a high correlation may be a situation in which the second test is partially correlated with the original test. I do not believe you can make comparisons on that basis unless you take account of partial correlations and particularly of the spurious correlations which may arise from the fractions.

Regarding Dr. Gregoire's statement that these tests are significantly different I should like to ask precisely what he means. If he used twice the standard error of the difference - I believe he did - he assumed a normal distribution curve and was using for some comparisons a fraction which I can

demonstrate was not normally distributed. I wish to ask also what he takes as a level of statistical significance when the distribution curve is skewed.

Dr. Wright and Dr. Gregoire, in presenting their data, showed the regression line with the tests scattered about it. That method of giving the data is much better, it seems to me, than the practice of expressing things as a percentage of the predicted normal. Instead of saying that somebody at that point is 100 per cent of a predicted normal, you should say that he lies within plus or minus a certain scatter of what would be expected for his age. In fact, you should subtract the average fall with age and not express it as a percentage. The expressing of it as a percentage assumes that it has a linear regression and that it goes about the mean - which it doesn't. If we are going to use statistics we should consult expert statisticians in order to avoid drawing erroneous conclusions.

I was extremely interested in Dr. Gregoire's conclusions about the differences between silicosis and asbestosis and I have no doubt that all he said was correct statistically from that point of view. I would like to ask whether he took cognizance of the various other individual tests, as far as their significant level was concerned.

DR. WRIGHT: Dr. Jones, I'm afraid you read more into my paper than I intended to have in it. You have gone beyond the limits of the paper and have applied the peak oxygen uptake to a man's ability to shovel coal. I refrained deliberately from doing that because I wished to discuss only what we measured. The factors related to a man's ability to perform his everyday tasks are most complex - his motivation, how many children he must feed - and why people become fatigued on their job is a most perplexing question. Why, in this era of labor-saving devices, is the housewife who may not have done much physical work

Just
but
not
in

often dead-tired at the end of the day? I cannot say, and I refrain deliberately from stating that the peak oxygen uptake will show whether or not an individual is able to carry out a full day's work. I can present many examples of men who break just as much rock underground as that broken by another man who may have double their peak oxygen uptake.

The peak oxygen uptake furnishes information on what the human engine is able to do on a precise piece of work.

(DR. WRIGHT MUST RE-WRITE)

etc etc

Old Stone Record page 531 -

Also how about other discussion - 535
by Dr McCann -

I find myself in full agreement with him on the question of the importance of the clinician and the tendency to rely on laboratory procedures. This reliance, in clinical medicine, is a manifestation partly of the tremendous burden that is put on a physician. I think it's also, to some extent, a false reliance in that we can see figures and understand figures more easily than we can the things that go into forming an opinion.

I do believe, however, that Doctor McCann would be the first to say that we need these gadgets and these methods of measurement if for no other reason than to apply some sort of a test to our clinical accounts, our clinical abilities.

The clinician has been going on for years expressing opinions, and lacking all - most always wanting to know, but lacking more precise methods of confirming his opinion, and I would think that perhaps one of the greatest functions - more precise measurement - is that of applying some sort of tests to clinical abilities, and it is somewhat in that vein that our work has been carried out and the study that I am to report this morning was done.

May I have the first slide? Because this is a mixed audience, I am going to take just a few moments to prepare some background for the comments that I am going to make, and I have hopes that my medical colleagues will forgive

me for this crude diagram which, to my mind, portrays in an easily understood manner, the vital functions of respiration.

The respiratory apparatus is in essence, a bellows, an air pump through which blood circulates for the purpose of being ventilated. Air enters into a bellows and the action of that bellows is all important. The air is then distributed through the bellows and being constantly replaced, maintains its way, with a higher pressure of oxygen and a lower pressure of carbon dioxide than existed in the blood that flows through it.

The venous blood returns to the lung from the periphery, carrying a high load of carbon dioxide and being low in oxygen. As the blood circulates through the bellows, gas passes across a very thick membrane which separates the blood phase from the gas phase of the lung, oxygen going into the blood and carbon dioxide coming out. The blood then returns to the left side of the heart and is pumped out through the tissues.

You have, in essence here, a pump and a diffusion service. That is the lung proper, and then we have, of course, a system that is intimately involved, it simply can not be separated from the lung, namely, the circulatory system, a pumping system which pumps the blood into the lung, accepts blood back from the lung and then pumps it out to the tissues.

Now, the vulnerable points of such an apparatus are, of course, immediately apparent. The apparatus is vulnerable from the point of view of air flow into and out of the lung, namely, from the pipes. Any airway obstruction will, very obviously, interfere with the capacity of this apparatus to behave as an air pump.

Improper distribution of gas in this bellows will lead to regional poor ventilation and possibly even -- actually it does occur -- areas of lung that are not ventilated at all. If these areas are still circulated, the venous blood that flows through such areas does so to no purpose whatsoever. The bellows itself is vulnerable. It is a pump in the same sense that any other mechanical pump operates. Loss of power or loss of deformability or ease of being changed in volume, will obviously impair its activity as a pump. The system is very vulnerable at that thin membrane which separates the blood from the gas phase. If it becomes thickened or otherwise changed, you can readily understand that gasses may have trouble in getting across this barrier and then, of course, the circulatory system itself is vulnerable, the total area which can receive blood or the cross-sectional area of the vascular bed may be diminished, so the blood is pumped through with difficulty. Blood may dam back in the lung, because of failure of the pump on the left side to move the blood along as it leaves

the lung. The circulatory aspect is going to be covered by Doctor McCann.

I don't, however, in our presentation, want you to lose sight of the fact that a lung is not just an air pump; it's intimately associated with the circulation.

Now, for a good many years, the - these intimate and distinct functions of respiration were not amenable to very precise study, but during the past twenty years, methods have been developed which will permit a fairly precise measurement of the activities of the respiratory apparatus especially the study of the vulnerable points that I have just mentioned.

I would like to say, at this point, that Doctor McCann and his group, Doctor Kotler, who is in the audience were and continued to be pioneers in this study. A really tremendous amount of the work that we are talking about today stems from it, their own laboratory, and I just think this is a rather appropriate time to recognize their role in studies of respiration in this country and abroad.

Now, some thirteen years ago, Doctor Gardner became interested in the functional aspects of the effects that the inhalation of dust might have on lung and circulation and it was because of this interest that I came here. I might say also that it gives me some pleasure to say that it was through Doctor McCann's efforts that this was made

possible. I doubt if many of you know that the initial building of the laboratory came about as a result of contributions for a number of years from the Department of Medicine, not only of money, but of time and helpful instruction. We began our studies in the only way that we knew how at the time, namely, to study the individual actions of the respiratory apparatus. These procedures are those that you have been told about here by others that have talked on the subject of pulmonary function.

We measured the size of the bellows, the so-called pulmonary volume; we measured the stroke volume of this air pump, then the vital capacity. We measured the power that this pump has to deliver air per minute, namely, the maximum breathing capacity. We have attempted, with relatively little success, to measure the distribution and mixing of gasses in the lung. With some greater success, we have been able to measure the impairment, if any, for the passage of gasses from the gas phase to the blood phase, and this is done in two ways, by studying the blood gasses of the blood after it has emerged from the lung, namely the degree to which the hemoglobin is saturated with oxygen and the degree to which carbon dioxide has been removed from the blood, and in a little more precise way, we have measured the pressure head that exists between the oxygen and carbon dioxide in the gas phase and the oxygen and carbon dioxide in the blood

phase.

One of the great shortcomings, and I think it is still a shortcoming, although a lot of work is being done on it, is that we have not been able to measure with nearly the same degree of accuracy, the role of the circulatory apparatus. I await with great eagerness the day when maximum cardiac outputs can be measured, and when the pressure relationships about which Doctor McCann will tell you, that govern the flow of blood through the lung, can be measured under conditions of stress.

After about four years of work, measuring these various things, these individual functions of the lung, we came to the realization that we were not really doing what we had set out to do. It is through, we could recognize, failure of the blood to be properly ventilated. We could recognize impairment of the pumping action of the lung or the respiratory apparatus. We could recognize poor distribution of gasses, and I have been guilty, as have others, of calling these disabilities, and thereby, we have confused some of our legal friends, because we have failed to be precise, and say these were disabilities for certain things.

A low maximum breathing capacity is a disability for breathing power. A poor arterial saturation is a disability for blood gas transfer and that is all that the disability is. That's all we have measured, and we and

others have sometimes not been careful to say whether or not this disability to breathing power is a disability in terms of a man's ability to work.

After, as I say, about four years of effort along these lines, we were repeatedly struck with the fact that certain men were remaining on the job doing a full day's work who had considerable impairment of breathing power, who had some degree of arterial unsaturation, who had abnormalities of the pulmonary volumena, and thick-headed as we were, took a long time to realize that we should be much more careful in the use of that word disability, and should define it precisely every time that it is used.

The word itself means exactly what it says, loss of an ability, and we should be careful to say what ability. Because of this discrepancy, we, as have many others before us, cast about for some way of trying to relate these specific disabilities to the general ability of a person to perform as a physical engineer - engine.

Now, you may use various procedures in this regard. The earning power of a man is the - to some extent, a measure of his ability to perform as a physical engine, but it is not a very precise one, and many factors are involved in that, perhaps I'll have occasion to say more about that this afternoon.

As doctors, we were, of course, interested in the

human body, and therefore, it is or would be nice if we could find some way of measuring the integrated man, what he can do in the way of physical performance. Some work has been done along these lines, measuring peak abilities to - for physical output, for example, the wonderful records that have been set in distance running and in other forms of physical feats, is, in a sense, a measure of an ability.

There is a more precise way of getting at it. The human engine is a great deal like an ordinary internal combustion engine. Fuel is burned, oxygen is required in that burning, and carbon dioxide is the result of that burning, and just as you might rate the power output of any engine in terms of its oxygen consumption, the human body can be studied in essentially the same way.

The next slide, please. One can study maximum performance abilities by having the individual walk on a treadmill rather than run on a circular one-mile track, and tremendous amounts of excellent work has been done in the Peck Laboratory at Harvard, studying fine athletes and some of that data I will show you in just a moment.

We have studied men from the point of view of their greatest ability to put out physical energy, by having them walk on a treadmill. We chose that rather than the bicycle because in this country, our people are not accustomed

to riding a bicycle. In addition, the exercise involved is not as free as it is in walking.

This is simply a picture of a man walking on a treadmill while various measurements are being made. Next slide. Studying the individual in this way, at increasing intensities of physical exertion, certain measurements can be made, as represented in this slide. This is of a person who has been walking or who has been walked at a constant speed, as you see, in the forward right-hand column, three and a half miles an hour, but increasing the level on which he walked until finally a level is reached which is the utmost that he can accomplish; in other words, a higher level, the walk is terminated at an earlier period than we have or had chosen.

These walks were all of six minutes duration and, by increasing the grade on which the man walks, we finally get to a point where the man can not walk for the full six minutes; he must get off and normal individuals, because of some severe leg weakness and some degree of breathlessness.

Now, it's apparent that the human engine is quite similar to the internal combustion engine. The oxygen uptake, as you would well expect, shown in the far left column, increases and gets up to a peak oxygen uptake of 2,950 cubic feet - cubic centimeters of oxygen. Now, that, then,

is the greatest ability that this individual had for oxygen uptake. If you would plot that out, you would see that as the last two exercises were carried out and a plateau was reached, there is very little increment in those last two exercises, and we have some data to show as in others, that a plateau is reached in oxygen uptake at a time when the minimum ventilation or breathing facility of the lungs can still be augmented and this has led along with other evidence, to the conclusion that in the normal man, the circulation is the governing factor for peak physical output of moderate duration. We're not talking now about short bursts of energy output.

I believe that I won't need to labor the point any further, but can simply say that it is generally accepted that the - one of the measures and perhaps one of the best at present available, of a man's ability to perform, as an integrated being, is the height to which he can increase or raise his oxygen uptake. This is certainly a function not only of respiration but of circulation, and I believe is primarily a function of circulation or chiefly so.

The other tables that I will show you, will indicate, as Doctor Motley stated, that we have enormous respiratory reserves, far greater than we do circulatory.

Now, we have chosen as our measure of the ability of this human engine to perform, this peak oxygen uptake,

and each man studied in the data that I will show you was carried in a series of walks of this sort to the peak oxygen uptake. The first two or three exercises can be done in one day. Subsequent ones, especially those requiring, when you get near the top, you can't do more than two in one day, because it's very strenuous work. It's very time-consuming, as you can see, so that what will be referred to as maximum oxygen uptake, is this bottom figure on the left-hand side, your left-hand side, and expressed in square meters of surface area, liters per minute, and what we have attempted to do or what we have accomplished in our study over the last ten years, is to run various tests of specific respiratory function in conjunction with this measurement of peak oxygen uptake.

Next slide. Now, here are forty-one normal men carried to the peak oxygen uptake on the treadmill. On the, on the left you see plotted on the orbit, the oxygen uptake in liters per square meter of body surface, and here the age in years. There is a loss of ability, as the man becomes older, which is only common sense, that fits in with what you all know.

The degree of spread is interesting in that I did anticipate there would be a greater variation. It is a good sized variation, standard deviation is approximately thirteen percent of the mean and that means that an individual

might -- well, I'll say more about that this afternoon -- really with normal mean bearing.

The next slide shows some abnormal individuals in respect to peak oxygen uptake. I might also say that the resting oxygen uptake is indicated, the amount of oxygen intake for a level walk is also indicated showing what reserves we possess. These are abnormal individuals and, to confirm the notion that peak oxygen uptake is a pretty good measure of a man's ability to perform, I have plotted data taken from a paper that appeared in Science, by Robinson Dill, and their co-workers, on peak power of physical performance of some of our fine athletes.

The very top figure is the data from Don Lasch, whom many of you will remember was our very fine two-mile runner, and very fine cross-country runner. The three that are plotted below him are Glen - Gene Benske, Glen Cunningham and Archie San Romani, who were also very fine distance runners. They have a perfectly phenomenal ability for oxygen uptake. Lasch could sustain that level not for five or six minutes but for twenty minutes.

Now, this is an ability that is far beyond anything that is possessed by the rest of us mere mortals. The average normal individual, as indicated by the very heavy line. These are some pathological subjects. The squares, filled in squares, are emphysemas. There is one

pneumonectomy over here, who still possesses a quite good ability. Some of the silicosis and asbestosis cases are scattered around.

Next slide. Now, I would like to show you a series of correlation coefficients in which we attempted to disclose or find out which of these specific measurements seem to parallel a man's greatest ability for oxygen uptake and, by inference, his ability to perform as a physical engine. The vital capacity correlated in a rather surprisingly good fashion. I have been one who, in the last ten years, has belabored the position of the vital capacity measurement. I'm not retracting all that I said about it, but I must say that this gave me some reason for surprise and I think Doctor Hugh-Jones, in his paper the other day, also said that they were somewhat surprised to find a way in which vital capacity correlated with ability of a man to perform.

This data, I must say, that I've got, is obtained on 180 individuals, and embraces all manner of pulmonary disease. There are sarcoid, there are pulmonary glaucomas, emphysema, bronchiectases, non-specific pneumoconiosis that we were able to diagnose, silicosis, asbestosis, exposure to dust, men with linear exposure and so on, a conglomerate group and ranges all the way from people who can just barely walk on the level at one and a half miles an hour for

five minutes, to men who had astounding abilities to perform, and it is, I believe, going to be possible with further working of our data to find out that there are specific diseases in which the vital capacity does give the erroneous information, and others in which the information is more reliable.

As you run down, you can see that there is very little improvement in the correlation when the vital capacity is expressed as a percent of the predicted, in other words, where you attempt to correct for age and surface area. Now, the residual capacity, or the residual air also correlates with a quite high correlation, as you would well expect, and as those have shown in the normal individuals, the correlation is very poor; in order to disclose the correlation, one must add in other people, namely, those who have had some impairment of their residual capacity.

It's rather interesting to show what Doctor Gregoire will later show, that in asbestosis, there is no correlation whatsoever.

Maximum breathing capacity correlates somewhat better than does the vital capacity.

The next slide. Splitting up the maximum breathing capacity, you see that in normal individuals, the correlation drops considerably, and I think this is further evidence that we possess far greater reserves for breathing

power than we do for circulation. If the maximum breathing capacity in normal men was a strong determinant of ability to perform, the correlation should be higher and I'm sure that when we are able to measure cardiac output during peak exercise, we will find a very high correlation. However, when you have impairment of the breathing pump as occurs in asbestosis and silicosis, the correlation is somewhat improved, especially in silicosis.

Next slide. The ability of the lung to remove carbon dioxide from the blood is, I think, one of the very important ones, and we were curious as to how that might correlate, and there is a correlation, but it is of a rather low order. Very poor correlation, as you see, between arterterial hemoglobin saturation during exercise - these were blood samples removed while the man was walking on the mill - very low correlation between any of the gas exchange measurements and peak ability to work.

I rather wish that Doctor Motley were here because he could correct me if I'm wrong in my impression that he left, that perhaps I'm wrong in this, because as I understand what he said, his belief is that the single best indication of whether a man is normal or not from a respiratory point of view, is whether his blood gasses are normal, and I think you see here a fairly convincing demonstration that a man's ability to work depends relatively little on

his arterial saturation.

This is not difficult to understand if one will recall the great civilizations have been built at altitudes where every one was unsaturated, and today some of our productive mines operate with some physical energy expended on the part of the men, at altitudes where everyone is to some extent, unsaturated. To be sure, compensations on the part of the body do occur, but the same thing is true in the diseased man, as Doctor McCann will undoubtedly describe to you, so that arterial blood gasses were surprisingly poorly correlated.

Now, the O_2V , that is a term that I must define for you, and could apply on one of the previous slides. It is what we refer to as the ventilation equivalent. It is obtained by dividing the minimum ventilation by oxygen uptake, and expressed, it's simply an expression of the breathing load, it's the amount of air that a man must breathe for a certain amount of energy expended, and it is surprisingly constant in normal people, has a very narrow range of variation.

Now, you see here a fairly high correlation, herein higher than we had anticipated, .60, and I shall jump now, just to the very last one, in which we had a multiple correlation. Incidentally, we had multiple correlations with most of these, and I'm only showing this one,

because it is the highest that we obtained. If one combines maximum breathing capacity and ventilation equivalent as a single factor, and I will show you some scattergraphs in a moment on that to point that out, then the correlation equivalent is a surprisingly high one, .904. This came as somewhat of a surprise to me. I still have difficulty in believing that it means what it seems to suggest, namely, that there is this very high dependence on M.B.C. and O₂V when one wishes to study peak abilities to perform.

On the other hand, it is somewhat common sense, isn't it, because maximum breathing capacity is, in essence, breathing power. Ventilation equivalent is, in essence, breathing load and the ability of an ordinary engine to perform is certainly directly related to the power of the engine and to the load that one puts on it.

Next slide. Now, we were interested in whether or not this correlation would hold up, of M. B. C. and O₂V versus oxygen uptake when one divided the data by diseases, and you will see that the correlation did hold up. Again, for normal individuals, the correlation is nowhere near that which we found in diseased individuals. This was another evidence that breathing reserves are much greater than circulatory reserves.

Next slide. Now, it's always well to look at scattergraphs, because as Doctor Hugh-Jones pointed out,

the correlation co-efficients usually look much better than do the scattergraphs, and the scatter will show you at once the wide variation which can also, of course, be expressed by the standard deviation which I will show you. This is vital capacity versus maximum oxygen uptake and there is, of course, this general slant, which is defined by the correlation co-efficient.

The standard deviation is large, as is shown by this wide scatter, making it, of course, very difficult to use the vital capacity as a good measurement in an individual, but for class studies or group studies, it is a better tool.

Next slide. This shows a maximum breathing capacity against peak oxygen uptake, and you will see at once that there is a - somewhat less scattered effect, still over large, and there are some exceptions that are always more interesting. Here, we find three individuals way down here all alone. These three persons were cases of pulmonary granuloma in beryllium workers, who possessed a normal maximum breathing capacity, but whose ability to exercise was very limited because of the very high ventilation equivalent, and when you correct the ventilation equivalent, they fall back into the body of the chart, as you will see in the next slide. Leave this one on just a moment.

You see, if you take above the average peak oxygen

uptake above 1.3, the scatter is tremendous, much more so than is true in the lower percentages here, indicating that respiratory reserves are far greater than circulatory or at least in relation to work. This shows you the M. B. C. and O₂V combined as a factor, and here you have a scatter that is certainly far less, again more scattered in the range of normal, because the M. B. C. has undue weight at this or in this portion of the distribution of our data. But here we have a quite nice distribution.

The next slide. As I say, it's one that surprised me. I throw this on primarily so that you can see the coefficient of variation which defines mathematically the degree of scatter around the regression line. Vital capacity twenty-one, 1,984 maximum breathing capacity and very great - very gratifyingly, much smaller for the combination of M. B. C. and O₂V.

The next slide brings you back again to the same distribution of M. B. C. over O₂V using, coming down now to zero instead of standing at three, with a drawing of a grid which combines the regression line and the standard deviation on the two sides.

The next slide -- or, that's all, I think. I want to summarize this rather long period of work by saying that as one would almost have anticipated, and I presume if I had gone to an engineer and said, "Now, look, we would

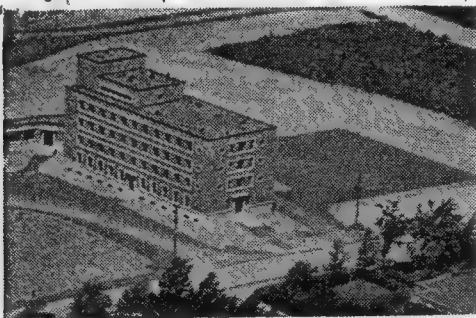
like to know what determines peak oxygen uptake", he would say, "Well, what's the load and what's the power", and we could have avoided all this work, because it's just a common sense correlation. I believe that the correlation is a rather surprising one. I can certainly say it's a surprising one to me. I don't understand why it's so high. I would think that circulation should have entered into this more than it did. We had no one in the series who had been in or was in congested failure. I'm sure we had some early cor pulmonales in the group. Undoubtedly, we have had some cardiac patients in this series, severe cardiacs, we would have found a little different result.

I believe that this type of study applied to the cardiac group would be very rewarding. In closing, I would like to say that this study is the end result of the labors of a large number of persons. I simply have the pleasure of presenting it to you. It's been carried on over the past ten years and the men who have worked in the laboratory have all taken a part in it. Thank you.

(Applause).

BY DOCTOR McCANN:

since the next speaker's material covers areas - much the same area, I think it would be better to defer the discussion on the two papers until we can have the two papers together, so I'll now ask Doctor Gregoire to present



LE BUREAU DU DIRECTEUR MÉDICAL

INSTITUT LAVOISIER
S O E U R S D E M I S É R I C O R D E
5757 BOULEVARD ROSEMONT, MONTRÉAL 36, P.Q., CANADA
TÉL. TURCOTTE 6961

Montreal, December 13, 1952.

Mrs. A.B. Blinn,
Saranac Laboratory,
P.O. Box 551,
Saranac Lake,
N.Y.

Dear Mrs. Blinn:

I am sending a copy of my conference to
Dr G. Wright who will make the corrections and will proba-
bly send it to you immediately after.

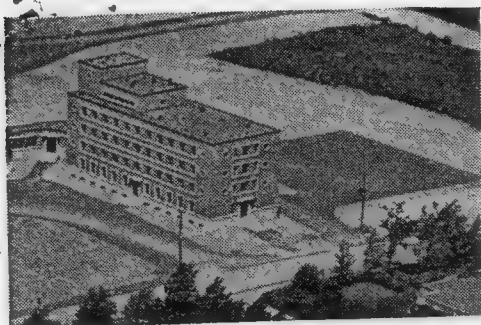
I hope there will not be too much delay
and at last you will know that I have sent this copy and
where you can get it.

With my best wishes for a Merry Christmas,

Very truly yours,

Dr. Fernand Grégoire, M.D.
Medical Director.

FG/fg



LE BUREAU DU DIRECTEUR MÉDICAL

INSTITUT LAVOISIER
S O E U R S D E M I S É R I C O R D E
5757 BOULEVARD ROSEMONT, MONTRÉAL 36, P.Q., CANADA
TÉL. TURCOTTE 6961

Montreal, December 9, 1952.

Mrs. Lillian R. Blinn,
Saranac Laboratory,
P.O. Box 551,
Saranac Lake,
N.Y.

Dear Mrs. Blinn:

Upon my arrival this morning I found your letter of December the 3rd concerning the report of the paper I gave to the Seventh Saranac Symposium in September. I asked my secretary to make a copy of this paper and to send it to you as soon as possible.

I also want to thank you for the informations you gave me concerning my Income Tax.

Very truly yours,

Dr Fernand Grégoire, M.D.
Medical Director.

FG/fg

December 3, 1952

Fernand Gregoire, M.D.
Medical Director
Institut Lavoisier
Socurs De Misericorde
5757 Boulevard Rosemont
Montreal 36, P. Q. Canada

Dear Doctor Gregoire:

The stenotypist's report of our Seventh Baranac Symposium has been received and we are attempting to pick up loose ends so that we may proceed with the editing. Your paper is the only one missing from our files and it so happened that the stenotypist's record did not include a transcript of your paper. Doctor Vorwald seems to be under the impression that you were going to send your formal paper to us. If you can let us have it within the next week or so we would certainly appreciate it.

Doctor Vorwald has not gotten around to thanking the speakers for their contributions to our sessions, but I know he plans to do this before very long. It was certainly a pleasure to have you with us.

Sincerely yours,

Lillian E. Blinn (Mrs. A.B.)
Executive Secretary to -
Arthur J. Vorwald, M.D.
Director

LHB:gn

December 3, 1952

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Institut Lavoisier
Socurs De Misericorde
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Director

LEB:ga

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Feb 3-1955

Original
sent to
DR Wright

Saranac Saranac Symposium

Saranac Lake, Saranac NY 1953

Sept. 25, 1952

PULMONARY FUNCTION STUDIES

IN MEN EXPOSED FOR TEN OR MORE YEARS TO

INHALATION OF ASBESTOS FIBRES

Fernand Gregoire*

and

George W. Wright

VORWALD COLL
BOX 91

26pp

From the Department of Physiology, The Trudeau Foundation,
Saranac Lake, New York

*Present Address:

Medical Director, Institut Lavoisier, Montreal, P. Q., Canada

Seventy-four men were subjects for this study, fifty-seven of whom had been, in the course of their daily work, exposed to an atmosphere containing airborne asbestos fibres in varying concentrations. The remaining seventeen men had no exposure to asbestos fibre other than that which might possibly occur to anyone living in their community. The purpose of this study was to learn what the physiological abnormalities are in persons with various degrees of pulmonary x-ray abnormalities and with various degrees of intensity of exposure to airborne asbestos fibre. It should be clearly understood that this study was not designed to disclose the proportion of men working in the asbestos industry who might have roentgenographic or physiologic abnormalities.

Now we can go to definitions.

How can one define and quantitate the various degrees of exposure to the inhalation of asbestos fibres? The thing to start with was the duration of exposure, as obtained from the subject and from the company. We arbitrarily assigned a value of two units for each year of exposure.

The various jobs at which the men worked were grouped in four categories on the basis of the intensity of exposure. This classification was arrived at after conference with the industrial engineers and plant physicians.

The men working underground or as loaders, drillers wet or in shipping shed, the primary crushers and dryers received one unit for their exposure.

The secondary crushers, the men working in dry rock storage or as dry drilling and grizzly were assigned three units. Floor men, screen men, millwright, tester, grader and cobber were assigned six units. The mill shipper, the bagger and the sewer were assigned nine units. Example:

A subject who spent 10 years as bagger had $10 \times 2 \times 9 = 180$ units and 10 years as millwright had $10 \times 2 \times 6 = 120$ units or a total of 300 units for 20 years of employment.

As far as the x-ray classification is concerned, we divided the cases in four groups on the basis of the degree by which they were abnormal.

We called the normal x-ray pattern 0. p^1 designated those who showed a slight exaggeration of linear markings. p^2 indicated a marked increase of the linear markings. p^{2+} categorized a haziness or ground glass appearance in the middle and basal fields of the lungs. This category in general embraced those films considered characteristic of clinical asbestosis.

It is difficult in asbestosis to be consistent with oneself in re-reading the films and still more difficult to agree with one another on each film. For this reason, each film was read twice by six different physicians--two radiologists, two plant physicians and two other clinicians. That made a total of 12 readings in each case. Usually there was no disagreement on extremes. The difficulty resided in the border line cases between p^2 and p^{2+} . These few cases were studied again until a final agreement was reached and where not, the weight of the decision was given to the radiologists.

In essence, we have attempted to rank the cases in respect to the degree to which the roentgenogram departs from normal.

Each person was thoroughly studied from the clinical standpoint as well as being subjected to the physiological measurements reported in the remainder of the discussion.

What are the abnormal physiological findings on a case who fits x-ray and clinical criteria of asbestosis?

The following patient is a good example of it.

Mr. W. F., 55 years old, had spent 20 years as bagger, 6 years in the mill and 8 years on surface where there was no dust. That gave him a figure of 430 units for duration X intensity of exposure. The x-ray that you see (Fig. 1) is quite characteristic of advanced asbestosis and this subject was unanimously called P^{2+} . For the last 16 years he was a chronic cougher, specially in winter, but expectorated little. He also complained of exertional dyspnea for the last 7 or 8 years.

The physiological data on him are as follows:

| | <u>Det.</u> | <u>Pred.</u> |
|--------------------|-------------|--------------|
| <u>M. B. C.</u> | 115 L/min. | 128 L/min. |
| <u>LUNG VOLUME</u> | <u>Det.</u> | <u>Pred.</u> |
| T. Vol. | 4.28 L. | 5.38 L. |
| V. C. | 2.84 | 4.06 |
| F.R.C. | 2.05 | 2.01 |
| Resp. Res. | 0.61 | 0.69 |
| Resid. C. | 1.44 | 1.32 |

ARTERIAL BLOOD AT EXERCISE: 3 mph. 4% grade 6 minutes

| | |
|--|-------------|
| O ₂ Cons./M ₂ /cc. | 635 cc. |
| O ₂ Content | 15.94 Vol.% |
| O ₂ Capacity | 20.70 Vol.% |
| % Saturation | 76 % |
| Alv. pO ₂ | 105 mm.Hg |
| Art. pO ₂ | 39 mm.Hg |
| A.A. Diff. | 66 mm.Hg |
| O ₂ v | 37 L. |

MAXIMUM EXERCISE: 3 mph. 8% 6 minutes

| | |
|-------------------------------------|-------------|
| Min. Vent. at 37° | 60.9 L/min. |
| O ₂ Cons./M ₂ | 775 cc. |
| O ₂ v | 43.4 L. |
| R. Q. | 1.03 |
| M.V./MBC | 0.54 |
| Resp. Rate (6th min.) | 40/min. |
| Pulse Rate (6th min.) | 168/min. |

The Maximum Breathing Capacity is 115 L/min. versus 128 L/min. predicted.

The Lung Volume showed a diminution of total volume. This is due to a great diminution of the vital capacity that is only 2.94 L. instead of 4.06 L. or 70% of the predicted values. The absolute figure for residual air is only slightly increased but in % over total volume it is increased to 34%. This figure alone,

however, cannot be considered abnormal since we have some subjects his age who were considered as normal and who showed the same percentage.

The data on the Arterial Blood during an exercise at 4% grade and 3 mph. are abnormal. The % saturation of hemoglobin is markedly subnormal during exercise. The alv. pO_2 in mm.Hg was absolutely normal at 105 mm.Hg. The art. pO_2 in mm.Hg was abnormally low at 39 mm.Hg compared to a mean normal of 82.5 with a S.D. of 5.41. The A.A. difference was 66 mm.Hg when the highest difference in normal persons at exercise is 27 mm.Hg.

At Maximum Exercise, 3 mph. and 8% grade, this patient hyperventilated considerably as can be seen from the high O_2 v. His O_2 consumption/ M_2 /min. attained only 775 cc. instead of 1285 as predicted for a normal man his age.

To summarize, the physiological abnormalities reside almost only in a diminution of the total volume of the lung, the unsaturation of the arterial blood at exercise, an increase of the A.A. difference due to a drop of arterial pO_2 and in the inability to perform as well as normals on the treadmill with hyperventilation.

Is it possible that one can get the typical x-ray changes of asbestosis, without the physiological abnormalities just seen, after a long time of exposure in a very dusty place?

Mr. D. L., 48 years old, is such a case. This subject worked one year as shipper, 20 years as bagger and two years as granite picker. This gave him a total of 366 units for duration X exposure. He was classified as P^{2+} (Fig. 2) and complained of

cough and slight exertional shortness of breath.

The Maximum Breathing Capacity was 71% of predicted or 100 L/min. instead of 140 L/min. Everybody who has less than 66% of predicted values is considered as abnormal because the S.D. of normals is 17%.

The Lung Volume was in normal limits, his total volume being 111% of the prediction and the residual air 28% of the total lung volume.

ARTERIAL BLOOD AT EXERCISE: 3.5 mph. 8% grade 6 minutes

| | |
|--|-------------|
| O ₂ Cons./M ₂ /cc. | 910 cc. |
| O ₂ Content | 19.86 Vol.% |
| O ₂ Capacity | 20.58 Vol.% |
| % Saturation | 96.5 % |
| Alv. pO ₂ | 98 mm.Hg |
| Art. pO ₂ | 78 mm.Hg |
| A.A. Diff. | 20 mm.Hg |
| O ₂ v | 26.9 L. |

As we can see, the saturation is perfectly normal and also the A.A. difference.

MAXIMUM EXERCISE: 3.5 mph. 12% grade 6 minutes

| | |
|-------------------------------------|-----------|
| Min. Vent. at 37° | 73 L/min. |
| O ₂ Cons./M ₂ | 1370 cc. |
| O ₂ v | 31.4 L. |
| R. Q. | 1.04 |
| M.V./MBC | 0.73 |
| Resp. Rate (6th min.) | 56/min. |
| Pulse Rate (6th min.) | 168/min. |

As you can see, all data are in normal limits. The O_2 consumed/ M_2 is even slightly greater than the predicted value for a normal man.

The question may next be asked, "Do physiologic abnormalities develop only after x-ray abnormalities are present?"

The next case illustrates the fact that in some instances there are physiological abnormalities in the absence of clear cut x-ray changes (Fig. 3).

Mr. A. S., 48 years old, worked as railway storekeeper from 1920 to 1935. From 1935 to 1948 he worked as fibre foreman in mills where the concentration of dust was considered as moderate, and the dust of a fine character. He received a total of 168 units for duration X intensity of exposure. The patient's only complaint was occasional head colds.

| | <u>Det.</u> | <u>Pred.</u> |
|-----------------|-------------|--------------|
| <u>M. B. C.</u> | 137 L/min. | 141 L/min. |

| <u>LUNG VOLUME</u> | <u>Det.</u> | <u>Pred.</u> |
|--------------------|-------------|--------------|
| T. Vol. | 5.38 L. | 5.28 L. |
| V. C. | 3.38 | 4.03 |
| F.R.C. | 2.53 | 1.98 |
| Resp. Res. | 0.53 | 0.73 |
| Resid. C. | 2.00 | 1.25 |
| % Resid. | 37% | 24.8% |

ARTERIAL BLOOD AT EXERCISE: 3.5 mph. 8% grade 6 minutes

| | |
|--|-------------|
| O ₂ Cons./M ₂ /cc. | 1100 cc. |
| O ₂ Content | 18.67 Vol.% |
| O ₂ Capacity | 20.87 Vol.% |
| % Saturation | 87.5 % |
| Alv. pO ₂ | 93 mm.Hg |
| Art. pO ₂ | 63 mm.Hg |
| A.A. Diff. | 30 mm.Hg |
| O ₂ ^v | 26.5 L. |
| R. Q. | .96 |

MAXIMUM EXERCISE: 3.5 mph. 16% grade 6 minutes

| | |
|-------------------------------------|-----------|
| Min. Vent. at 37° | 77 L/min. |
| O ₂ Cons./M ₂ | 1385 cc. |
| O ₂ ^v | 29.5 L. |
| R. Q. | 1.07 |
| M.V./MPC | 0.56 |
| Resp. Rate (6th min.) | 30/min. |
| Pulse Rate (6th min.) | 172/min. |

The Maximum Breathing Capacity is normal.

There was a slight increase of the % residual air due to a diminution of the vital capacity and to an increase of the residual air in liters; however, 37% is a border-line figure.

The Arterial Blood shows a significant diminution of the saturation and a diminution of the arterial pO₂ with an increase of the A.A. difference to 30 mm.Hg.

It is interesting to note that this relative unsaturation did not influence the capacity of this man for work when he is compared to the group of so called normal subjects his age. He consumes even a little more O_2/M_2 than the mean predicted value and at 3.5 mph. and 16% grade he does not hyperventilate much, his O_{2v} being only 29.5 L.

One can also raise the question as to whether or not all persons subjected to prolonged heavy exposure will of necessity develop abnormal x-ray and physiological changes. This is not necessarily so as demonstrated by the next case.

Mr. L. L., 48 years old, spent 28 years in a very dusty place, the shipping department as shipper and shipping foreman in mills. This gave him a total of 504 units for duration and intensity of exposure. His x-rays (Fig. 4) show no evidence of asbestosis and he was classified O. He had no complaints.

| | <u>Det.</u> | <u>Pred.</u> |
|-----------------|-------------|--------------|
| <u>M. B. C.</u> | 158 L/min. | 139 L/min. |

| <u>LUNG VOLUME</u> | <u>Det.</u> | <u>Pred.</u> |
|--------------------|-------------|--------------|
| T. Vol. | 6.57 L. | 6.57 L. |
| V. C. | 4.74 | 4.94 |
| F.R.C. | 2.59 | 2.47 |
| Resp. Res. | 0.76 | 0.86 |
| Resid. C. | 1.83 | 1.76 |
| % Resid./T. Vol. | 28% | 24.8% |

ARTERIAL BLOOD AT EXERCISE: 3.5 mph. 10% grade 6 minutes

| | |
|--|--------------|
| O ₂ Cons./M ₂ /cc. | 1105 cc. |
| O ₂ Content | 19.79 Vol. % |
| O ₂ Capacity | 21.73 Vol. % |
| % Saturation | 91.1 % |
| Alv. pO ₂ | 93 mm.Hg |
| Art. pO ₂ | 74 mm.Hg |
| A.A. Diff. | 19 mm.Hg |
| O ₂ ^v | 24.1 L. |
| R. Q. | .83 |

MAXIMUM EXERCISE: 3.5 mph. 12% grade 6 minutes

| | |
|-------------------------------------|-------------|
| Min. Vent. at 37° | 85.9 L/min. |
| O ₂ Cons./M ₂ | 1475 cc. |
| O ₂ ^v | 28.0 L. |
| R. Q. | 1.12 |
| M.V./MBC | 0.55 |
| Resp. Rate (6th min.) | 30/min. |
| Pulse Rate (6th min.) | 160/min. |

The Maximum Breathing Capacity and the Lung Volume are absolutely normal. The blood data are also normal and the O₂ consumption in cc./M₂ at maximum exercise is higher than the mean normal.

Now going to group study. It should be re-emphasized that the men we have studied do not include all of the men exposed in any one plant nor, in addition, are these men random samples of the working population. The men were chosen on the basis that

they had the degree of exposure needed for the study, that they be free of extraneous disease (diabetes, etc.) and that they be willing to cooperate. Since, however, there was no deliberate bias, it will be profitable to consider certain groups with the reservation that our data may possibly not indicate the true proportion of abnormality that exists in the industry as a whole. Using the data that we have, we may proceed to certain questions.

First question: Does it exist a correlation between the duration and intensity of exposure and the development of x-ray patterns characteristic of asbestosis? Table I is self-explanatory.

Table I. EXPOSURE RATING VS X-RAY CLASSIFICATION

| Units Characterizing Exposure | 0 | p ¹ | p ² | p ²⁺ | |
|-------------------------------|----------------|----------------|----------------|-----------------|------------------|
| 17 had not been exposed 0 | 11 | 5 | 1 | - | = 17 |
| 3 had between 0 and 100 | 1 | 1 | 1 | - | = 3 |
| 5 had between 100 and 200 | - | 2 | 3 | - | = 5 |
| 23 had between 200 and 300 | 5 | 3 | 7 | 8 | = 23 |
| 19 had between 300 and 400 | 4 | 2 | 4 | 9 | = 19 |
| 7 had more than 400 | $\frac{1}{22}$ | $\frac{1}{14}$ | $\frac{2}{18}$ | $\frac{3}{20}$ | = $\frac{7}{74}$ |

There is no good correlation. Out of 23 cases who had 200 to 300 units, five or 20% are classified 0 and three P¹ still considered as normal. Eight or 35% were true asbestosis cases. About the same distribution is seen for exposure to higher concentrations. It is of interest that there were no severe x-ray changes in persons whose exposure rating was less than 200.

For the purposes of correlating exposure with various physiologic measurements, we have used only the men over age 40,

thus excluding the small group of seven men below that age. Table II shows that when the group is divided on the basis of severity of exposure, the age distribution is homogenous and hence can be neglected in the remainder of the tables related to exposure.

Table II. EXPOSURE. Age over 40

| | Mean | S.D. | S.E. | Δ | Significance |
|---------|------|------|------|----------|--------------|
| Normal | 50.9 | 5.23 | 1.09 | | |
| 0 | 50.7 | 6.78 | 1.75 | .2 | None |
| 200-300 | 49.2 | 6.91 | 1.51 | 1.7 | None |
| 300-400 | 48 | 6.58 | 1.64 | 2.9 | None |
| 400+ | 51.4 | 4.47 | 1.69 | .5 | None |

Table III. EXPOSURE VS M.B.C. Det./L/Min.

| | Mean | S.D. | S.E. | Δ | S.E. Δ | Significance |
|---------|-------|-------|-------|----------|---------------|--------------|
| Normal | 124.9 | 15.84 | 3.3 | | | |
| 0 | 128 | 15.44 | 3.99 | 3.1 | 5.18 | No S. |
| 200-300 | 111.5 | 23.34 | 5.09 | 13.4 | 6.07 | S. |
| 300-400 | 101.7 | 20.99 | 5.20 | 23.2 | 6.16 | S. |
| 400+ | 107.3 | 29.58 | 11.19 | 17.6 | 11.67 | No S. |

If we study the data, we can see that the mean is lower and significantly so specially for the subjects in the group 300 to 400, but some individual, are still in normal limits and the group of 400 and over has a higher figure than the preceding group, possibly due to the fact that the number of subjects is only 7 in this group.

Table IV. EXPOSURE VS M.B.C. AS % OF PREDICTED

| | Mean | S.D. | S.E. | Δ | S.E. Δ | Significance |
|---------|------|-------|------|----------|---------------|--------------|
| Normal | 100 | 17% | | | | |
| 0 | 94.5 | 13.9 | 3.60 | 1.6 | 4.6 | No S. |
| 200-300 | 81.4 | 15.64 | 3.41 | 11.5 | 4.57 | S. |
| 300-400 | 72.2 | 13.13 | 3.28 | 20.7 | 4.47 | S. |
| 400+ | 80.1 | 21.62 | 8.17 | 12.8 | 8.72 | No S. |

In % prediction, the same is true.

Tables III and IV disclose that there is a perceptible diminution of the Maximum Breathing Capacity in the exposed group but the reduction does not appear to bear any relationship to intensity and duration of exposure.

Table V. EXPOSURE VS TOTAL VOLUME OF LUNG

| | Mean | S.D. | S.E. | Δ | S.E. Δ | Significance |
|---------|------|-------|------|----------|---------------|--------------|
| Normal | 5.93 | .97 | .20 | | | |
| 0 | 6.06 | .97 | .25 | .13 | .32 | No S. |
| 200-300 | 4.67 | .995 | .25 | 1.26 | .30 | S. |
| 300-400 | 4.76 | 1.028 | .26 | 1.17 | .33 | S. |
| 400+ | 5.03 | .98 | .37 | .90 | .42 | S. |

Table VI. EXPOSURE VS VITAL CAPACITY

| | Mean | S.D. | S.E. | Δ | S.E. Δ | Significance |
|---------|------|------|------|----------|---------------|--------------|
| Normal | 4.12 | .63 | .13 | | | |
| 0 | 4.22 | .65 | .167 | .10 | .21 | No S. |
| 200-300 | 3.08 | .778 | .17 | 1.04 | .214 | S. |
| 300-400 | 2.96 | .647 | .16 | 1.16 | .21 | S. |
| 400+ | 3.22 | .83 | .313 | .90 | .34 | S. |

Tables V and VI disclose a definite restriction of the size to which the lung can be enlarged on full inspiration and of stroke volume in those persons exposed to asbestos fibre, but again there is no relationship between the magnitude of the change and the severity of exposure.

Table VII. EXPOSURE VS RESIDUAL AIR ABSOLUTE/L.

| | Mean | S.D. | S.E. | Δ | S.E. Δ | Significance |
|---------|------|------|------|----------|---------------|--------------|
| Normal | 1.81 | .466 | .097 | | | |
| 0 | 1.84 | .475 | .123 | .03 | .16 | None |
| 200-300 | 1.59 | .356 | .078 | .22 | .124 | None |
| 300-400 | 1.80 | .55 | .14 | .01 | .17 | None |
| 400+ | 1.80 | .49 | .18 | .01 | .20 | None |

Table VII shows that there is no interference with the degree to which the lungs can be emptied.

Table VIII. EXPOSURE VS ARTERIAL BLOOD GASES DURING EXERCISE

| | 0 | 0-100 | 100- 200 | 200- 300 | 300- 400 | 400+ |
|-------------------------------------|------|-------|-------------|-------------|-------------|------|
| No. of Persons Studied | 17 | 3 | 5 | 23 | 19 | 7 |
| Mean % Hb. Sat. with O ₂ | 94.9 | 93.4 | 88.2 | 90.2 | 91.3 | 92.4 |
| No. of Abnormal Persons | 0 | 0 | 3 | 10 | 7 | 1 |
| A.A. O ₂ Difference | 20.9 | 23.0 | 31.5 | 31.4 | 27.3 | 30.1 |
| No. of Abnormal Persons | 0 | 0 | 3 | 12 | 6 | 2 |

Table VIII discloses that of the 57 exposed persons, approximately two-fifths evidenced definite impediment to the passage of O₂ from the alveoli into the pulmonary capillary blood. No ready explanation is available for the fact that there seems to be a more severe disturbance in this regard among those persons with less severe exposure as compared to those more severely exposed.

Table IX. MAXIMUM ATTAINABLE O₂ UPTAKE DURING EXERCISE

| | 0 | 200- 300 | 300- 400 | 400+ |
|---|------|-------------|-------------|------|
| Maximum O ₂ Uptake as % of Predicted for Normal | 102% | 86% | 85% | 80% |
| No. of Persons Falling Below 2 S.D. from Prediction | 0 | 4 | 5 | 3 |

Each man was walked on the treadmill for six minutes at successively greater intensities of work until the maximum ability to increase O₂ uptake was reached. In a normal group previously studied in this laboratory, this ability was shown to correlate highly with age, thus permitting one to predict what value a

normal person should attain with a coefficient of variation equal to 13.8%. Table IX discloses the relationship between asbestos exposure and capacity for maximum O_2 uptake (energy expenditure) in the men studied. Twelve of the persons were found to be distinctly subnormal and there does appear to be a slight but not good correlation with intensity of exposure.

To summarize, there is a striking lack of demonstrable correlation between the development of physiologic abnormalities and the severity of exposure to asbestos fibre as rated on the basis of duration and intensity of exposure.

A similar comparison of physiologic data and x-ray classification in an attempt to discover correlations was made. For this purpose we omitted the small group of seven men under 40 years of age. Table X shows a homogeneous distribution among the various categories of roentgenogram appearance as regards age; hence, the effect of age alone can be disregarded.

Table X. X-RAY CLASSIFICATION. Age over 40

| | No. of Cases | Mean | S.D. | S.E. | Δ | Significance |
|--------|--------------|------|------|------|----------|--------------|
| Normal | | 50.9 | 5.23 | 1.09 | | |
| 0 | 22 | 48.2 | 7.25 | 1.66 | 2.7 | None |
| p1 | 14 | 50.2 | 5.74 | 1.99 | .7 | None |
| p2 | 16 | 48.7 | 6.69 | 1.73 | 2.2 | None |
| p2+ | 22 | 51.0 | 5.35 | 1.33 | 1.1 | NONE |

Tables XI and XII show that the Maximum Breathing Capacity is significantly low both in absolute quantity and as % of predicted normal values in categories p² and p²⁺ but there is no apparent difference between the degree of change in Maximum Breathing Capacity of these two categories.

Table XI. X-RAY CLASSIFICATION VS M.B.C. Det./L/Min.

| | Mean | S.D. | S.E. | △ | Significance |
|--------|-------|-------|------|------|--------------|
| Normal | 124.9 | 15.84 | 3.30 | | |
| 0 | 124.3 | 20.79 | 4.77 | .6 | No S. |
| p1 | 112.2 | 27.52 | 7.63 | 12.7 | No S. |
| p2 | 107.4 | 26.48 | 6.84 | 17.5 | S. |
| p2+ | 106.5 | 19.46 | 4.35 | 18.4 | S. |

Table XII. X-RAY CLASSIFICATION VS M.B.C. AS % PREDICTED

| | Mean | S.D. | S.E. | △ | Significance |
|--------|------|-------|------|------|--------------|
| Normal | 100 | 17% | | | |
| 0 | 89.8 | 18.61 | 4.27 | 10.2 | No S. |
| p1 | 82.6 | 17.88 | 4.96 | 17.4 | No S. |
| p2 | 77.4 | 18.74 | 4.84 | 22.6 | S. |
| p2+ | 79.8 | 12.20 | 2.73 | 20.2 | S. |

Inspection of Table XIII shows a striking diminution of Total Volume, especially in those showing more severe roentgenographic change. That this reduction is due primarily to a diminution in the Vital Capacity is clearly shown in Table XIV.

Table XIII. X-RAY CLASSIFICATION VS TOTAL VOLUME DET.

| | Mean | S.D. | S.E. | Δ | Significance |
|--------|---------|------|------|----------|--------------|
| Normal | 5.93 L. | .97 | .20 | | |
| 0 | 5.66 | .927 | .213 | .27 | No S. |
| p1 | 5.81 | .882 | .245 | .12 | No S. |
| p2 | 4.91 | .826 | .213 | 1.02 | S. |
| p2+ | 4.10 | .759 | .170 | 1.83 | S. |

Table XIV. X-RAY CLASSIFICATION VS VITAL CAPACITY DET.

| | Mean | S.D. | S.E. | Δ | Significance |
|--------|---------|------|------|----------|--------------|
| Normal | 4.12 L. | .63 | .13 | | |
| 0 | 3.89 | .675 | .155 | .23 | No S. |
| p1 | 3.88 | .744 | .206 | .24 | No S. |
| p2 | 3.14 | .627 | .162 | .98 | S. |
| p2+ | 2.62 | .489 | .109 | 1.50 | S. |

The residual air in absolute values shows no significant change except for a slight reduction as displayed in Table XV. As is shown in Table XVI there is a significant increase in the residual air expressed as a % of Total Volume but this is the result of a drop in Vital Capacity alone and in no way caused by an increase of the residual air.

Table XV. X-RAY CLASSIFICATION VS RESIDUAL AIR ABSOLUTE

| | Mean | S.D. | S.E. | Δ | Significance |
|--------|------|------|------|----------|--------------|
| Normal | 1.81 | .466 | .097 | | |
| 0 | 1.77 | .428 | .098 | .04 | No S. |
| p1 | 1.93 | .43 | .119 | .12 | No S. |
| p2 | 1.77 | .489 | .126 | .04 | No S. |
| p2+ | 1.50 | .428 | .096 | .31 | S. |

Table XVI. X-RAY CLASSIFICATION VS RESIDUAL AIR AS % OF T.V.

| | Mean | S.D. | S.E. | Δ | Significance |
|--------|------|-------|-------|----------|--------------|
| Normal | 30.4 | 4.40 | 0.92 | | |
| 0 | 31.3 | 5.34 | 1.23 | .9 | No S. |
| p1 | 33.6 | 6.397 | 1.774 | 3.2 | No S. |
| p2 | 35.9 | 7.85 | 2.03 | 5.5 | S. |
| p2+ | 36.1 | 5.135 | 1.15 | 5.7 | S. |

These data on pulmonary volumina are of interest for two reasons. The histologists report the occurrence of "emphysema" as a prominent coincident lesion with asbestosis. In clinically significant diffuse obstructive emphysema, the residual air is always enlarged in absolute values. In our series, there is no evidence of an increase in residual air. This fact, considered together with the fact that the Maximum Breathing Capacity is only moderately reduced even in severe p2+ cases is strong evidence that diffuse obstructive emphysema of a clinically significant degree is not a prominent or important feature of the functional aberrations attending asbestosis. Actually, the characteristic effect of

asbestosis on the pulmonary dynamics and volumina is to cause a restriction of lung mobility--well characterized as a "tight" or "restricted" lung. Inspection of the pulmonary volumina data strongly suggests that the abnormalities parallel the severity of x-ray abnormality.

The data shown in Table XVII shows that interference with the passage of O_2 across the alveolar membrane was not at all uncommon and occurred with increasingly great frequency as the changes in the roentgenogram were more pronounced. It is important to point out that the majority but not all of those categorized $p2^+$ (frank asbestosis) exhibited some abnormal degree of impediment to O_2 transfer. Of perhaps even greater interest is that some of those persons whose roentgenograms were not sufficiently abnormal to warrant a classification of $p2^+$ (frank asbestosis) also showed clearly recognizable abnormal impediment to O_2 transfer. This finding suggests that the roentgenogram is not necessarily the earliest and most sensitive indicator of injury caused by inhalation of asbestos fibre.

Table XVII. ARTERIAL BLOOD GAS STUDIES VS X-RAY CHANGE

Arterial Saturation During Exercise

| X-ray Category | Normal | 0 | p1 | p2 | p2+ |
|--------------------|--------|------|-------|------|------|
| No. of Cases | 30 | 22 | 14 | 16 | 22 |
| Mean % Saturation | 94.95 | 94.1 | 93.56 | 93.1 | 87.5 |
| S. D. | 2.33 | 2.52 | 3.63 | 4.5 | 6.64 |
| No. Abnormal Cases | None | None | 2 | 4 | 15 |

Table XVII. (Continued)

Alveolar-Arterial O_2 Difference During Exercise

| | | | | | |
|--------------------|------|------|------|------|-------|
| Mean in mm.Hg | 19.6 | 20.1 | 22.0 | 27.0 | 30.0 |
| S. D. | 4.74 | 5.8 | 6.89 | 7.28 | 10.93 |
| No. Abnormal Cases | None | None | 3 | 5 | 15 |

In Table XVIII is shown the relationship between the maximum capacity for O_2 uptake during physical work of six minutes' duration and the pattern of the roentgenogram. There is a significant decrease in the P² and P³⁺ groups but it is important to note that the majority of these two groups still retained normal capabilities for maximum O_2 uptake. This observation fits well with clinical experience. Although the data will not be shown until a subsequent publication, our evidence clearly shows a lack of any correlation between the occurrence or severity of arterial blood gas abnormality during exercise and the ability of the person to reach a high level of O_2 uptake during physical exercise.

Table XVIII. O_2 CONSUMPTION AT PEAK EXERCISE VS X-RAY CHANGE O_2 Cons./ M_2 /cc./min. at Peak of Exercise in % of Predicted

| X-ray Category | Normal | 0 | P ¹ | P ² | P ³⁺ |
|--------------------|--------|-------|----------------|----------------|-----------------|
| No. of Cases | 36 | 22 | 14 | 16 | 22 |
| Mean | 100 | 99.98 | 96.42 | 86.11 | 78.25 |
| S. D. | 13.8 | 10.87 | 11.46 | 13.18 | 13.20 |
| No. Abnormal Cases | None | None | None | 2 | 9 |

Of the total group of 74 subjects in this study, 17 had never been exposed to asbestos fibre and in the sub-group no physiological abnormality was found. In the remaining 57 subjects, all of whom were occupationally exposed to asbestos fibre, physiological abnormalities were observed with the following frequencies.

| | |
|---|-------------|
| Subnormal Arterial pO_2 during exercise | 26 subjects |
| Abnormally increased Alveolar-arterial O_2 difference during exercise | 23 subjects |
| Subnormal Vital Capacity | 22 subjects |
| Arterial hemoglobin unsaturation during exercise | 21 subjects |
| Subnormal maximum ability for O_2 uptake during exercise | 18 subjects |
| Subnormal Maximum Breathing Capacity as % of predicted normal value | 9 subjects |

Summary

This study dealt with 74 men of whom 57 cases had been subjected for many years to different concentrations of asbestos dust.

It was found that there was some but not good correlation between the duration and the intensity of exposure and the development of the x-ray pattern characteristic of asbestosis. Insofar as abnormal physiological data are concerned, a better correlation is found with the x-ray pattern than with intensity and duration of exposure.

The most frequent abnormalities were found to be a diminution of the arterial pO_2 and an increase of the A-A difference during exercise; a diminution of the vital capacity; and a diminution of the percentage of saturation of the arterial blood during exercise.

There is no true correlation, however, between these findings and the diminution of the capacity of a subject to work on a treadmill. A man can still be well saturated and unable to perform normally, while another one can be unsaturated and able to consume as much O_2/M_2 as the mean of normals his age.

This study shows that the physiologic abnormality is chiefly that of a "tight" lung that is expanded with difficulty and impairment of gas transfer between the gas and blood phases of the lung.

This is in rather striking contrast to the abnormality that is most commonly seen in silicosis, namely obstructive emphysema with its impairment of ventilation.

APPENDIXMeaning of Abbreviations Used

| | |
|------------|----------------------------|
| M.B.C. | Maximum Breathing Capacity |
| Det. | Determined |
| Pred. | Predicted |
| T. Vol. | Total Volume |
| V. C. | Vital Capacity |
| F.R.C. | Functional Residual Air |
| Resp. Res. | Respiratory Reserve |
| Resid. C. | Residual Capacity |

BLOOD AT EXERCISE

| | |
|--|--|
| 3 mph. | 3 miles per hour |
| 4% | 4 inches of slope per 100 inches length |
| O ₂ Cons./M ₂ /cc. | Oxygen consumed per square meter of body surface are in cc. per minute |
| Alv. pO ₂ mm.Hg | Pressure of oxygen in the alveoli in millimeters of mercury |
| Art. pO ₂ | Pressure of oxygen in the arterial blood |
| A.A. Diff. | Difference of pressure in mm.Hg between the alveolar and the arterial oxygen |
| O ₂ ^v | Oxygen equivalent = the number of liters of air ventilated to consume one liter of oxygen. |

GREGOIRE

APPENDIX (Continued)

S. D.

Standard Deviation

Min. Vent. at 37°

Ventilation in liters per minute at

37° centigrade

R. Q.

Respiratory Quotient

his paper on the pulmonary function studies in men exposed for ten or more years to the inhalation of asbestos fibers.

BY DOCTOR GREGOIRE:

(Doctor Gregoire read a prepared paper which was to have been filed with the Saranac Laboratory. Transcript of his remarks is available in the event a formal paper was not filed).

BY DOCTOR McCANN:

There may be considerable discussion of these two papers, but I suggest that we take about a four or five minute recess for a stretch before we discuss them.

(Recess from 12:30 to 12:40 P. M.).

BY DOCTOR VORWALD:

May we all take our seats?

BY DOCTOR McCANN:

Will the meeting please come to order? Will you please come to order? Will all those who have not gotten their banquet tickets please do so before twelve o'clock, before twelve. The two papers which I have just heard by Doctor Wright and by Doctor Gregoire are now open for discussion.

BY DOCTOR HUGH-JONES:

I would like to make two comments really. The first is about Doctor Wright's paper with which I was extremely interested, and he put forward a suggested definition,

shall we say, of a way of measuring a man's capacity for overall work. There was one question I would like to ask him about that, and that is this, that many people, I think I'm right in saying, Taylor and Briggs originally did that which they called the crest load, the maximum work that people can do in a steady state. Other people have done the maximum ability to work as measured by the oxygen consumption which Doctor Wright has used.

Now, there is always one thing that worries me slightly about that, and that is this, that although you and I, for instance, might have very different powers to do our maximum, does that necessarily limit us at the ordinary work we're going to do in every day performance. In fact, the question is this, that does Doctor Wright have evidence to show that the performance at sub-maximal loads is necessarily worse in people who show a falling off of their maximum ability? It doesn't always seem to me to necessarily follow.

For example, I might have a reduced maximum ventilation or maximum ventilatory power of my lungs and, therefore, not be able to run, shall we say, a half a mile as fast as I could before. On the other hand, I might be able to walk at a slow rate for a long time perfectly well if my own thinking on that subject, and I would like his comments on that.

The other thing I want, and I hope people will excuse me because I'm not a statistician, I'm a male physiologist, and I have a certain amount of fears, and worries about statistics. I think you know about the same kind of known estimations that are given that if all the blood capillaries were put end to end, they would stretch around the world. Somebody once said if all the statisticians were put end to end, that would be a good thing.

With those remarks, if I may make two technical criticisms on the statistics, and I hope you'll forgive me. The first is on the use of this correlation co-efficient. Doctor Wright was surprised at the extremely high co-efficient he got between the maximum breathing capacity combined with the ventilation equivalent for oxygen, and the maximum power to work. I believe that is an example of what is technically called a spurious correlation. He has got the same thing coming into the top and bottom of the fraction, namely, the ventilation in maximum breathing capacity in one case and in ventilation equivalent on the other, and I believe in his examples of comparing correlations, he should have been using partial correlations throughout, that if you are comparing the overall correlation, what you're observing is the amount one test relates to some other test, and the degree of that is a set thing if you're just comparing one set of tests. If you're comparing

another test to the first thing, in this case oxygen capacity for work, the reason why that correlates so highly may be due because it's partially correlated with your original test and I don't think you can put out or compare things on that basis unless you take a count of the partial correlations and particularly of the spurious correlations which may arise from fractions.

That is one technical point. There are two others. One is that it does seem to me that if people, and I would like to ask Doctor Gregoire this question, when he says that these tests are significantly different, what he precisely means. If he uses twice the standard error of the difference which I think he did, that assumes a normal distribution curve and in a lot of these things which he was comparing, he is using a fraction which I know and can demonstrate is not normally distributed.

I would like to ask him what he takes as a level of statistical significance when the thing is on askewed distribution, and then lastly, which I think is an important point, and I put it up in all humility because I'm not sure the real answer to it, and that is this, that is a great fashion in physiology to which many people have been addicted, and that is to express things in a percentage of the predicted normal.

Now, it does seem to me that what Doctor Wright

and Doctor Gregoire did in two of their slides was a much more valuable way of doing things, and that is they showed the regression line with these tests scattering about it, and I think instead of saying that somebody at that point is a hundred percent of a predicted normal, you should say he lies within plus or minus a certain scatter of what would be expected at that age. In fact, you should subtract the average fall with age and not express it as a percentage. To express it as a percentage assumes, for one thing, a linear regression. It assumes the thing goes about the mean which it doesn't in any case, and lastly, in the extremes, it is entirely incorrect and it's a thing in physiology, I think we ought to fight very seriously, that if we are going to use statistics, I think we ought to consult expert statisticians on those things, and I do make those comments in all humility because I do think a lot of the physiological conclusions can be erroneous.

I was extremely interested in Doctor Gregoire's conclusions, his last ones, about the differences between asbestosis and silicosis, and I have no doubt in my own mind that all he said was extremely correct statistically from that point of view, and I think it was a fascinating and a very valuable and able study though I would like to ask him whether he did take cognizance of the various other individual tests as far as their significance level was

concerned.

BY DOCTOR McCANN:

We're going to finish by your scheduled time, it will be necessary to limit this discussion and I'll ask Doctor Wright if he cares to answer Doctor Hugh-Jones.

BY DOCTOR WRIGHT:

Doctor Jones, I'm afraid you read more into my paper than I wanted to be in it. You have gone beyond now and are applying the peak oxygen uptake to a man's ability to shovel coal, and I have refrained from doing that deliberately, because this is what we measured, and that's all I want to talk about, what enters into a man's ability to perform every day his most complex; motivation alone, how many children he must feed at home is extremely important, why people become fatigued on their job is a most complex question. Why does the housewife who really, in many instances, has little physical labor to do, with our washing machines and so on, become dead tired at the end of the day? I wouldn't for a moment say, and I refrain deliberately from saying that peak oxygen uptake will tell you whether or not the man is able to carry out a full day's work.

I can show you, I'm sure you have many examples, of men that stay on the payroll. They break just as much tonnage underground as the next man who may have double his

peak oxygen uptake.

What I do believe the peak oxygen uptake gives you some information on is what his human engine is able to do in this precise piece of work.

Now, there are two extremes that this sort of falls in the middle of. There is the sudden burst of energy which a man may be called upon to do in running from a burning building, which a severe cardiac might be able to do. That's accomplished anaphobia. Then there is the other kind that you are referring to, the sub-maximal work that is carried out over a long period of time and that may not bear a relationship to the peak oxygen uptake.

As a matter of fact, it was the failure of its bearing relationship that led us to do this study originally, because we had observed just in a practical way, that men were -- with a low maximum breathing capacity who had arterial saturation, who had a high residual of air, were nevertheless able to do a full day underground, doing a job that was certainly a sub-maximal job, so I agree with you fully. I'm glad you brought the point up that one can not transfer this sort of data in any exact manner to the question of whether or not an individual is able to do a day's work at sub-maximal labor.

I do believe, however, that it is a tool that lets the clinician find out how well he is able to estimate the

departure from normal.

Now, our statistical problem would take a considerable amount of time. I recognize the things you are talking about and would like to have more opportunity to converse with you in that regard. We were worried about whether or not we had the same background, two sides of the correlation, a thing that I believe was a serious criticism of Doctor Motley's data.

It's true that oxygen, oxygen uptake, is a factor that appears, and ventilation appears, but they are done under different circumstances and I do not believe they are the same.

It would be something like saying that a man who has one dollar is a millionaire because he possesses a dollar. Oxygen uptake, during work, when it's related to ventilation equivalent, is simply the amount of oxygen being used in relation to the amount of air being breathed at the time. It's totally different from maximum breathing capacity and it's totally different from peak oxygen uptake and I think we're on fairly safe grounds from that point of view.

You raise the question of askewed distribution. It's true that one of these correlations definitely is a curvo-linear distribution and that's the maximum breathing capacity against maximum oxygen uptake. The curve is in the

process of being fitted at the present time in an effort to see whether it would give us a better correlation than the formula we did use, which was a straight line formula.

I would like to say one thing that Doctor Gregoire perhaps won't be able to touch on, and that is when we say a thing is a certain percent of the predicted, we are taking into consideration the two standard deviations from normal. These are most of the time corrected curves for age or some other factor and the standard deviation of the normal predicted value has been calculated as, for example, with maximum breathing capacity in which we say there is such a figure as one hundred percent or a normal figure, the scatter around that regression line gives you a standard deviation if you wish to calculate it.

The scatter, for example, around the maximum breathing capacity, is such that ties the standard deviation of the predicted value requires you to consider all people over sixty-six percent as being within the normal range. Now, the matter of partial correlation is much too involved for us to discuss here. We can do that afterwards.

BY DOCTOR McCANN:

Do you wish to make further comment, Doctor Gregoire?

BY DOCTOR GREGOIRE:

I think he has covered it.

BY DOCTOR McCANN:

If not, I will proceed then to discuss the subject which was assigned to me, which concerned the cardio-circulatory aspects of Pneumoconiosis.

I might say at the outset in answer to a remark of Doctor Wright's, that I do not think the clinician should omit functional and physiological studies. The function of the clinician is to put the whole mess of things together. He's an integrator. He must take all aspects of the man concerned, into account, if it's going to make any sense.

(Doctor McCann's paper, from this point, was prepared, and a copy has been filed with the Saranac Laboratory.

BY DOCTOR McCANN:

In the printed record of these proceedings, I should like the names of Doctor Lovejoy, Hillfrank and others as co-authors of the paper which I have read. I'm afraid I haven't been too clear. I certainly have used up so much time, I'll certainly leave very little for discussion.

Doctor Waring will open the discussion.

BY DOCTOR WARING:

Doctor McCann and Members of the Symposium, Doctor McCann has, as always, presented factual matter as well as philosophical comments with such a happy facility that I envy him. I'm reminded of the story which I hope our British friends here will not find incorrect, about Sir Joseph

CURRENT RESEARCHES IN ANESTHESIA & ANALGESIA



Official Organ of the International Anesthesia Research Society

LAURETTE McMECHAN, Ass't. Editor, EXECUTIVE SECRETARY
318 HOTEL WESTLAKE, ROCKY RIVER, CLEVELAND 16, OHIO

February 3, 1953

Dr. James J. Waring
University of Colorado
Department of Medicine
4200 East 9th St.,
Denver 7, Colo.

Dear Dr. Waring:-

We are happy to give you permission to use the two illustrations from Drs. Whitehead and Draper's paper, namely numbers 1 and 4, with due acknowledgement to source of the original publication.

With kindest personal regards, I am,

Cordially and appreciatively yours,

Laurette McMechan
Assistant Secretary

Chapter ^{Twenty-six} Twenty-five

Cardio-circulatory Aspects of the Pneumoconioses

William S. McCann, M.D.

Call this
Chap 26

The mechanisms controlling.....
.....(Dr. McCann's paper on file).....
.....within the lung.

THE UNIVERSITY OF ROCHESTER
SCHOOL OF MEDICINE AND DENTISTRY
AND
STRONG MEMORIAL HOSPITAL
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PHYSICIAN-IN-CHIEF

OCTOBER 1, 1952

DR. ARTHUR VORWALD
SARANAC LABORATORY
SARANAC LAKE, N. Y.

DEAR ARTHUR:

DO YOU WANT CUTS OF THE SLIDES USED FOR THIS PAPER?

IF NOT, HERE IT IS READY FOR PRINTING.

WITH KIND REGARDS.

SINCERELY YOURS,

William S. McCann
WILLIAM S. McCANN, M.D.

WSM/w

CARDIO-CIRCULATORY ASPECTS OF THE PNEUMOCONIOSES

WILLIAM S. McCANN, M.D.*

(WITH THE COLLABORATION OF
FRANK W. LOVEJOY, JR. AND PAUL N. G. YU)**

ROCHESTER, N. Y.

See page 535 of Steno record
Hilfman
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* FROM THE DEPARTMENT OF MEDICINE, UNIVERSITY OF
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** AIDED BY A GRANT FROM THE UNITED STATES PUBLIC
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TO BE READ BEFORE THE 7TH SILICOSIS SYMPOSIUM AT
SARANAC LAKE, N. Y. THURSDAY, SEPTEMBER 25, 1952.

THE MECHANISMS CONTROLLING PULMONARY VENTILATION AND CIRCULATION ARE SO INTIMATELY RELATED THAT IT IS VIRTUALLY IMPOSSIBLE TO CONSIDER ONE WITHOUT THE OTHER, EITHER IN NORMAL OR PATHOLOGICAL INDIVIDUALS. IT IS ALSO TRUE THAT NO CONSIDERATIONS OF THE PULMONARY AND CARDIAC MECHANISMS HAVE VALIDITY UNLESS THEY INCLUDE THE ENTIRE CIRCULATION AND RESPIRATION, PERIPHERAL AS WELL AS PULMONIC, AND THE CENTRAL AND VEGETATIVE NERVOUS INTEGRATION OF THESE FUNCTIONS.

IT HAS LONG BEEN REALIZED THAT IN SOME INSTANCES ADVANCED PATHOLOGICAL CHANGES IN THE LUNGS RESULT IN FAILURE OF THE RIGHT VENTRICLE OF THE HEART. THIS HAS BEEN DESIGNATED AS PULMONARY HEART DISEASE, OR COR PULMONALE, WHEN THIS EVENT OCCURS AS A RESULT OF GREATLY INCREASED PULMONARY RESISTANCE TO THE TRANSFER OF BLOOD FROM THE RIGHT VENTRICLE TO THE LEFT. THE SITUATIONS RESULTING IN FAILURE OF THE RIGHT VENTRICLE TO ADAPT TO INCREASED PULMONARY RESISTANCE MAY ARISE ACUTELY, OR MAY RESULT FROM REACHING THE CRITICAL LIMITS IN SLOWLY DEVELOPING LONG COMPENSATED PATHOLOGICAL CONDITIONS IN THE LUNGS. WE HAVE RECOGNIZED ACUTE COR PULMONALE FOLLOWING EXTENSIVE EMBOLIZATION OF THE LUNGS, AND IN ANAPHYLACTIC SHOCK. THE CHRONIC COR PULMONALE, WITH WHICH WE ARE CONCERNED TODAY, IS THE HEART FAILURE OF HYPOVENTILATED EMPHYSEMA, OR OF EMPHYSEMA PLUS FIBROSIS.

PRIOR TO THE LAST WAR OUR CONCEPTS OF COR PULMONALE, WERE DOMINATED BY PURELY MECHANICAL THINKING, BASED ON OUR KNOWLEDGE OF THE MORBID ANATOMICAL CHANGES IN THE LESSER CIRCULATION ACCOMPANYING CHRONIC PULMONARY DISEASE. SINCE THE WAR THE ASPECTS OF THIS PROBLEM HAVE BEEN ENTIRELY CHANGED BY A FLOOD OF NEW LIGHT ON THE SUBJECT. THE KEY TO THESE NEW DEVELOPMENTS WAS THE INTRODUCTION OF THE CARDIAC CATHETER BY Cournand and Ranges (5).

AS SOON AS THEIR TECHNIQUES WERE APPLIED TO THE STUDY OF INTACT MAN IT BECAME POSSIBLE, NOT ONLY TO STUDY THE PRESSURE CHANGES IN THE RIGHT SIDE OF THE HEART, PULMONIC ARTERY AND PULMONARY CAPILLARIES, BUT ALSO THE RESPIRATORY GAS TENSIONS IN THE MIXED VENOUS BLOOD, TO CALCULATE PULMONARY BLOOD FLOW BY THE FICK PRINCIPLE, AND TO DESIGN MANY CLEVER EXPERIMENTS FOR CORRELATING THE ADAPTIVE FUNCTIONS OF THE RESPIRATION AND CIRCULATION. THE RESULT HAS BEEN A BRILLIANT ILLUMINATION OF THE WHOLE PROBLEM, AND A SHIFT OF INTEREST FROM PURELY MECHANICAL EVENTS TO THE CHEMICAL, HORMONAL, AND NERVOUS MECHANISMS UNDERLYING THEM. IN THE IMMEDIATE PHASE OF THIS DEVELOPMENT WE HAVE STILL CENTERED OUR INTEREST ON EVENTS OCCURRING WITHIN THE THORAX, BUT WE ARE SHOWING SIGNS OF APPRECIATING THE PRACTICAL SIGNIFICANCE OF ADVANCES IN NEUROPHYSIOLOGY, NOT ONLY OF THE RESPIRATORY CENTER AS IT MAY BE

AFFECTED BY CEREBRAL BLOOD FLOW, BUT OF THE NUMEROUS PERIPHERALLY SITUATED PRESSO- AND CHEMORECEPTORS, KNOWLEDGE OF WHICH HAS BEEN SO WELL REVIEWED BY HEYMANNS ^{18/}✓ AND PI-SUÑER ^{36/}✓, WHICH GREATLY EXTEND WHAT WE FORMERLY KNEW ABOUT THE AORTIC AND CAROTID MECHANISMS.

THIS WHOLE DEVELOPMENT ILLUSTRATES BEAUTIFULLY THE DIRECTION OF CHANGE IN CLINICAL MEDICINE. IT IS NO LONGER POSSIBLE TO SOLVE ITS PROBLEMS IN THE LIGHT OF KNOWLEDGE OF THE ANATOMICAL AND PHYSIOLOGICAL CHANGES IN SINGLE ORGANS AND SYSTEMS. WE REALIZE, AS NEVER BEFORE, HOW MUCH EACH CELL DEPENDS UPON EVERY OTHER CELL, THE WHOLE ADDING UP TO THE "PERSONALITY" OF THE ORGANISM. FOR MANY YEARS THE PENDULUM SWUNG IN THE DIRECTION OF MORE AND MORE DIFFERENTIATION AND SPECIALIZATION IN THE PURSUIT OF KNOWLEDGE; NOW IT IS SWINGING THE OTHER WAY TOWARD INTEGRATION AND UNDERSTANDING OF THE ADAPTIVE RESPONSES OF THE WHOLE PERSON TO HIS WHOLE ENVIRONMENT.

IN 1946 LAUSON, BLOOMFIELD, AND CURNAND ^{22/}✓ PUBLISHED A STUDY OF THE EFFECT OF RESPIRATION ON CIRCULATION IN NORMAL MEN. THE SLIDE WHICH I WILL SHOW IS TAKEN FROM THEIR PAPER.

SLIDE 1

IN THIS SCHEMA IT WILL BE NOTICED THAT THE INTRA-THORACIC PRESSURE DECREASES TO A MINIMUM AT THE END OF INSPIRATION

OF MINUS 8 CM. H_2O , THE MEAN PULMONARY ARTERIAL PRESSURE RISES AND RIGHT VENTRICULAR STROKE VOLUME ^{rise} TO A MAXIMUM AT THE SAME POINT, WHILE THE LEFT VENTRICULAR STROKE VOLUME AND MEAN SYSTEMIC ARTERIAL PRESSURE DECREASE.

ONE OBSERVES THAT THE RIGHT AND LEFT VENTRICULAR OUTPUTS VARY IN RECIPROCAL DIRECTIONS DURING A SINGLE RESPIRATION; THE DIFFERENCE BETWEEN THEM IS REFLECTED IN THE CHANGING VOLUME OF BLOOD WITHIN THE LUNGS. FROM THIS WE MAY INFER THAT IN PULMONARY HYPERTENSION THESE RECIPROCAL DIVERGENCES MIGHT INCREASE THE DIFFERENCE IN OUTPUT OF THE TWO VENTRICLES EVEN TO THE POINT AT WHICH STARLING'S LAW OF THE HEART WOULD HAVE TO BE APPLIED TO EACH VENTRICLE SEPARATELY (McCANN ²⁵ ~~28~~ ✓)

SLIDE 2

IN THIS FIGURE ARE SHOWN THE SIMULTANEOUS AND RECIPROCAL VARIATIONS IN PRESSURE IN THE THORACIC AND ABDOMINAL PORTIONS OF THE VENA CAVA DURING A SINGLE INSPIRATION AND EXPIRATION. THIS IS TAKEN FROM AN EXPERIMENT OF HERBERT R. BROWN, JR., IN OUR LABORATORIES.

THIS ILLUSTRATES GRAPHICALLY ONE OF THE MAJOR MECHANISMS FOR RETURNING VENOUS BLOOD TO THE HEART, AND FOR COORDINATING THE BLOOD FLOW WITH THE VENTILATION OF THE LUNGS. ONE CAN READILY SEE THAT THIS MECHANISM WOULD BE DISTURBED PROFOUNDLY BY

SUCH ABNORMALITIES ON THE ABDOMINAL SIDE AS RELAXATION OF THE MUSCULATURE, DIASTASIS OF RECTI, ABDOMINAL HERNIAS, AND VARI-COSITIES OF THE VEINS OF THE LEGS, AND ON THE THORACIC SIDE BY ADHESIONS OR EFFUSIONS IN PLEURA OR PERICARDIUM, AND BY ANYTHING WHICH TENDS TO ELEVATE THE ALVEOLAR AIR PRESSURE, AS IN BREATH HOLDING DURING ASCENT FROM A DIVE, POSITIVE PRESSURE BREATHING, OBSTRUCTION OF THE AIR PASSAGES, THE VALSALVA MANEUVER, SEVERE ASTHMA, ANAPHYLACTIC SHOCK, AND EVEN PAROXYSMAL COUGH. WE WERE ABLE TO RECORD AN INSTANCE OF THE LATTER CONDITION TO WHICH WE GAVE THE NAME "TUSSIVE SYNCOPÉ"²¹, IN WHICH THE RIGHT VENTRICULAR PRESSURE ROSE DURING A BOUT OF COUGHING TO MORE THAN 200 MM. HG., AND INTERRUPTED THE CIRCULATION SO THAT SYNCOPÉ AND CONVULSIONS OCCURRED. THIS CONDITION HAD BEEN DESCRIBED BY CHARCOT IN 1876 AND NAMED BY HIM "LARYNGEAL EPILEPSY".

SLIDE 3

NEXT I WOULD LIKE TO SHOW YOU A CRUDE SCHEMA OF THE TOTAL CIRCULATION IN ORDER TO BRING OUT CLEARLY CERTAIN BASIC RELATIONSHIPS WITHIN THE CIRCULATION.

SLIDE 4

THE MOST BASIC FACT TO BE BORNE IN MIND AT ALL TIMES IS STARLING'S "LAW OF THE HEART", WHICH STATES: "THE ENERGY SET FREE AT EACH CONTRACTION OF THE HEART IS A SIMPLE FUNCTION OF

THE LENGTH OF THE FIBERS COMPOSING ITS MUSCULAR WALL."

IN OTHER WORDS THE OUTPUT OF THE HEART IS TO A LARGE EXTENT DETERMINED BY THE VOLUME AND PRESSURE OF THE VENOUS INPUT.

DURING DIASTOLE THE RIGHT AURICLE AND VENTRICLE ARE DISTENDED BY THE INCOMING TIDE OF VENOUS BLOOD, AND THE FORCE OF THE CONTRACTION OF THE RIGHT VENTRICLE IS DETERMINED THEREBY.

UNDER NORMAL CONDITIONS THE DISCHARGE OF THE RIGHT VENTRICLE DETERMINES THE DEGREE OF DIASTOLIC DISTENSION OF THE LEFT HEART AND THUS THE INPUT AND OUTPUT OF THE TWO BALANCE.

UNDER ABNORMAL CONDITIONS, AS WE SHALL SEE LATER, INTRATHORACIC CHANGES MAY IMPEDE THE TRANSFER OF BLOOD FROM THE RIGHT VENTRICLE TO THE LEFT SO THAT THE OUTPUT OF THE LEFT VENTRICLE IS DIMINISHED AND THE BALANCE OF THE TWO VENTRICLES IS UPSET.

BEFORE WE PURSUE THIS SUBJECT FURTHER I WOULD LIKE TO DIRECT YOUR ATTENTION TO THE RELATIONSHIP OF THE BRONCHIAL AND CORONARY CIRCULATIONS TO THE PULMONIC. THE FIRST TWO ARE IN THE REVERSE DIRECTION TO THE PULMONIC, CARRYING BLOOD FROM LEFT VENTRICLE TO THE RIGHT.

THE BRONCHIAL CIRCULATION ANASTOMOSES WITH THE PULMONIC (AND WITH THE CORONARY AS WELL). THE NATURE OF THE BRONCHIAL-PULMONIC INTER-RELATIONSHIP WAS CLEARLY APPRECIATED BY VIRCHOW⁴² IN 1850, WHEN HE POINTED OUT THAT THE BRONCHIAL

FURNISHED COLLATERAL CIRCULATION WHEN THE PULMONIC WAS BLOCKED, AND THAT THE BRONCHIAL VESSELS BECOME ENLARGED IN VARIOUS TYPES OF CHRONIC PULMONARY DISEASE. THESE WISE OBSERVATIONS HAVE BEEN CONFIRMED IN RECENT YEARS BY SCHLAEPFER ³⁹✓ AND MORE RECENTLY BY WOOD AND MILLER ⁴⁴✓, AND LIEBOW AND LINDSKOG ²⁵✓.

IN THE YOUNG THE COMMUNICATIONS BETWEEN THESE TWO ARTERIAL SYSTEMS IS LITTLE IN EVIDENCE, BUT AS AGE PROGRESSES AND PULMONARY AND CARDIAC STRESSES MULTIPLY THEY BECOME MORE AND MORE EASY TO FIND. IN OUTSPOKEN PULMONARY AND CARDIAC DISEASE THEY BECOME MANIFEST. THROUGH A BRONCHOSCOPE ONE MAY SEE VARICES OF THE BRONCHIAL VEINS IN PATIENTS WITH MITRAL STENOSIS, AS SHOWN BY FERGUSON, KOBILAK, AND DIETRICH ¹³✓.

I THINK WE MAY ASSUME FROM THESE FACTS, THAT WHEN THE RESISTANCE TO TRANSFER OF BLOOD FROM THE RIGHT VENTRICLE TO THE LEFT IS INCREASED, WHETHER THIS RESISTANCE BEGINS IN THE PULMONIC CIRCUIT OR WHETHER IT BEGINS AT THE MITRAL VALVE, THERE WILL PROBABLY BE A CERTAIN PORTION OF THE BLOOD DISCHARGED FROM THE RIGHT VENTRICLE RETURNED THROUGH THE BRONCHIAL VEINS TO THE RIGHT AURICLE. AS PRESSURE IN THE RIGHT AURICLE RISES, STASIS IN THE BRONCHIAL VENOUS SYSTEM MIGHT BE A FACTOR IN PULMONARY CONGESTION AND EDEMA. THIS SAME FACTOR APPLIES TO THE LYMPHATIC SYSTEM, DRAINAGE OF WHICH CAN BE AFFECTED ONLY ADVERSELY BY RISING PRESSURE IN THE GREAT VEINS. IT SHOULD BE REMEMBERED THAT WILLIAM SNOW MILLER ³³✓ AND ROUVIERE ³⁸✓ HAVE

SHOWN THAT THE LYMPHATICS OF THE LUNG EXTEND DOWN THE BRONCHI ONLY TO THE RESPIRATORY BRONCHIOLES, AND THAT THERE ARE NONE IN THE ALVEOLAR WALLS.

WITH THIS FIGURE STILL BEFORE YOU LET US CONSIDER THE PROBLEMS OF CONGESTIVE FAILURE OF THE CIRCULATION. IN THE LABORATORIES OF CARL WIGGERS ATTEMPTS WERE MADE BY LEVY AND BERNE ²³✓ TO PRODUCE CONGESTIVE FAILURE EXPERIMENTALLY. THESE WORKERS TRIED NUMEROUS MEANS OF DOING THIS BY INJURIES TO THE LEFT VENTRICLE, BY TRAUMA, CORONARY LIGATIONS, TOXINS, ET CETERA. ALL OF THESE EFFORTS FAILED TO PRODUCE CONGESTIVE FAILURE. SUCCESS WAS ATTAINED BY ONE METHOD ONLY, THAT OF APPLYING CONSTRICTION TO THE MAIN PULMONARY ARTERY.

CONGESTIVE FAILURE IS CHARACTERIZED BY RISING PRESSURES IN THE RIGHT HEART AND VEINS, AND DIMINISHED OUTPUT OF THE LEFT VENTRICLE. THIS IS WELL SHOWN IN NUMEROUS PAPERS BY McMICHAEL AND OTHERS ³¹✓. CONSIDERED MECHANICALLY, IN THE LIGHT OF THIS DIAGRAM, CONGESTIVE FAILURE CAN BE BROUGHT ABOUT IN ONLY TWO WAYS:

- 1) OVER-DISTENSION OF THE RIGHT HEART BY A FLOOD OF INPOURING VENOUS BLOOD, AND
- 2) INCREASED RESISTANCE WITHIN THE LUNG TO THE TRANSFER OF BLOOD FROM THE RIGHT VENTRICLE TO THE LEFT SO THAT THE OUTPUT OF THE LEFT VENTRICLE IS REDUCED.

McMICHAEL ³¹✓ HAS RECOGNIZED THESE TWO TYPES UNDER THE DESIGNATION OF "HIGH OUTPUT" AND "LOW OUTPUT" FAILURES, AND HAS ILLUSTRATED THE OPERATION OF STARLING'S LAW IN THESE CASES IN THE FOLLOWING FIGURE.

SLIDE 5

IN THIS DIAGRAM YOU WILL OBSERVE THE NORMAL OPERATION OF STARLING'S LAW UPON A HEALTHY HEART IN THE UPPERMOST CURVE, ON WHICH THE CROSS ILLUSTRATES THE VENOUS FILLING PRESSURE AND CARDIAC OUTPUT AT REST, AT A POINT WELL BELOW THE MAXIMUM WHICH THE NORMAL HEART CAN ATTAIN.

IN THE MIDDLE CURVE THE MAXIMUM OUTPUT WHICH THE HEART CAN ATTAIN IS LESS THAN NORMAL AND THE RESTING VENOUS PRESSURE ~~REQUIRED TO ATTAIN IT~~ IS HIGHER THAN NORMAL.

IN THE LOWER CURVE THE RESTING VENOUS PRESSURE IS HIGHER THAN THAT AT WHICH A LOW MAXIMUM OUTPUT CAN BE ATTAINED.

THE CONDITIONS UNDERLYING "HIGH OUTPUT FAILURES" ARE SUCH AS THESE:

A) ARTERIO VENOUS ANEURYSMS

B) BERI-BERI

C) SEVERE ANEMIAS

D) HYPERTHYROIDISM

E) PAGET'S OSTEITIS

F) EMPHYSEMA, to which will probably be added

G) CHRONIC CO₂ INTOXICATION

IN ALL OF THESE CONDITIONS THE TRANSFER OF BLOOD FROM ARTERIES TO VEINS MAY BE INCREASED, BOTH AS TO RATE AND VOLUME, SO THAT THE RIGHT VENTRICLE IS OVER DISTENDED BY THE INCOMING FLOOD OF VENOUS BLOOD TO THE POINT OF FAILURE, *when it is no longer adequately protected by the hepatic "check valve".*

THE CONDITIONS UNDERLYING "LOW OUTPUT" FAILURE ARE THOSE IN WHICH THE CAPACITY OF THE LEFT VENTRICLE TO INCREASE ITS OUTPUT SUFFICIENTLY TO MEET THE METABOLIC DEMANDS FOR OXYGEN IS IMPAIRED, SUCH AS:

- A) ISCHEMIA OF LEFT VENTRICULAR MUSCLE
- B) HYPERTENSION
- C) LEFT SIDED VALVULAR LESION
- D) CONSTRICTIVE PERICARDITIS

ET CETERA

ALL ATTEMPTS TO EXPLAIN THESE TWO TYPES OF CONGESTIVE FAILURE WHICH ARE BASED ON PURELY MECHANICAL OR ANATOMICAL FEATURES ARE DOOMED TO FAILURE. WEAKNESS OR INJURY TO THE LEFT VENTRICLE DOES NOT SIMPLY BACK UP BLOOD IN THE LUNGS. THE FIRST PHENOMENA FOLLOWING A CORONARY OCCLUSION IN THE LEFT VENTRICLE ARE NOT THOSE OF CONGESTIVE FAILURE, BUT ARE THOSE OF SHOCK. IF CONGESTIVE FAILURE ULTIMATELY RESULTS FROM CORONARY OCCLUSION, IT IS A SECONDARY EVENT. I SHALL ATTEMPT TO DEVELOP AN HYPOTHESIS TO EXPLAIN WHY THIS IS SO.

LET US SUPPOSE THAT A WEAKENED LEFT VENTRICLE IS FACED WITH A STRESS IN WHICH IT IS UNABLE TO PUT OUT ENOUGH BLOOD TO

MAINTAIN THE NORMAL OXYGEN DEMAND OF CELLS OF THE BODY, SO THAT AN OXYGEN DEFICIENCY OCCURS.

THE FIRST REACTION TO OXYGEN LACK, BEFORE COMPENSATION SETS IN IS HYPERVENTILATION WHICH LEADS TO A LOSS OF CARBON DIOXIDE. THIS IS WELL ILLUSTRATED BY THE BEHAVIOUR OF AN UNACCLIMATIZED MAN ON FIRST ASCENT TO A HIGH ALTITUDE.

SLIDE 6

THE FIGURE SHOWN IS TAKEN FROM DILL'S BOOK, "LIFE, HEAT AND ALTITUDE", SHOWING THE DATA OF H. T. EDWARDS ⁷✓. THIS FIGURE SHOWS CLEARLY THE DIMINUTION OF CO₂ TENSION (PCO₂) AND CO₂ CONTENT OF THE ARTERIAL BLOOD AS ALTITUDE INCREASES. PARTIAL COMPENSATION OCCURS, BUT THE pH OF THE ARTERIAL BLOOD IS SHIFTED DEFINITELY TO THE ALKALINE SIDE (FROM pH 7.35 TO 7.46). HYPOCAPNIA RESULTING FROM HYPERVENTILATION IS ASSOCIATED WITH DIMINISHED BLOOD FLOW THROUGH THE BRAIN AS SHOWN BY KETY, SCHMIDT, AND OTHERS /, AND ALSO BY PERIPHERAL CONSTRICTION WHICH TENDS TO DELAY AND DIMINISH THE TRANSFER OF BLOOD FROM ARTERIES TO VEINS. BY REDUCING THE VENOUS RETURN THE CARDIAC OUTPUT IS LIMITED.

IT IS PROBABLE THAT THE STATE OF SHOCK WHICH CHARACTERISTICALLY OCCURS FOLLOWING AN INFARCTION IN THE LEFT VENTRICLE IS, AT LEAST IN PART, TO BE EXPLAINED BY THIS MECHANISM.

THIS IS ILLUSTRATED BY A PATIENT OBSERVED ON OUR WARDS RECENTLY. THIS PATIENT HAD PROLONGED HYPOTENSION FOLLOWING A CORONARY OCCLUSION. WHILE BREATHING ROOM AIR HIS ARTERIAL O_2 SATURATION GREATLY REDUCED, HIS PCO_2 WAS 40 MM. HG. AND THE PH WAS 7.48. WHEN HE WAS GIVEN 100 PER CENT OXYGEN TO BREATHE THE ARTERIAL OXYGEN SATURATION ROSE, THE PCO_2 ALSO ROSE FROM 40 TO 52 MM. HG. AND THE PH FELL TO 7.38. AS THE PCO_2 ROSE THE BLOOD PRESSURE AND PULSE VOLUME MARKEDLY IMPROVED.

TABLE 1.

CO_2 TENSION AND SHOCK FOLLOWING MYOCARDIAL INFARCTION

| BREATHING | PCO_2 MM.HG. | PO_2 MM.HG. | CO_2 CONTENT MM/L. | O_2 CONTENT VOL. % | O_2 SAT. % | PH |
|------------|-------------------|------------------|-------------------------|-------------------------|-----------------|------|
| Room Air | 40 | 44 | 21.7 | 13.08 | 71.5 | 7.48 |
| 100% O_2 | 52 | 81 | 23.5 | 17.46 | 95.6 | 7.38 |

IF WE EXAMINE THE DATA OF PETERS AND BARR ²⁸✓, WE FIND THAT THE ARTERIAL PCO_2 OF PATIENTS WITH HEART FAILURE RANGED BETWEEN 39.7 - 52.2 MM.HG. THESE VALUES RANGE FROM NORMAL TO SLIGHT ELEVATION, AND ARE QUITE LOW COMPARED WITH VALUES OF 80 - 90 MM. HG. AND HIGHER USUALLY SEEN IN CASES OF PULMONARY FAILURE.

IN INTERPRETING THESE RESULTS WE HAVE POSTULATED THAT, IN THE DEVELOPMENT OF PRIMARY HEART FAILURE, ANOXIC HYPERVENTIL-

ATION THREATENS THE HOMEOSTASIS OF CO_2 AND BLOOD pH , AND THAT AN ADAPTIVE REACTION OCCURS IN THE LUNGS WHICH MAY PROTECT THE BODY AGAINST CO_2 LOSS OR HYPOCAPNIA. IN SUPPORT OF THIS POSTULATE IT CAN BE SAID THAT MEASUREMENTS OF THE LUNG VOLUME IN CONGESTIVE FAILURE CHARACTERISTICALLY SHOW A DECREASE IN THE VITAL CAPACITY AND INCREASE IN RESIDUAL AIR (IN THE ABSENCE OF PLEURAL EFFUSION). THIS CHANGE IS SUCH AS WOULD DIMINISH ALVEOLAR VENTILATION AND LIMIT CO_2 LOSS.

IN EXPERIMENTS ON NORMAL MEN, WHICH WERE CARRIED OUT ON OURSELVES (HURTADO, KALTREIDER, AND McCANN ¹⁹✓) IN A LOW PRESSURE CHAMBER, WE FOUND THAT A SIMILAR DECREASE IN VITAL CAPACITY AND RISE IN RESIDUAL AIR OCCURRED. THIS, AS WE WILL SHOW LATER, IS PROBABLY THE ADAPTIVE RESPONSE IN THE LUNGS TO THE NEED FOR CONSERVING CO_2 .

PROGRESSIVE FAILURE OF THE LUNG - GENESIS OF COR PULMONALE.

THE MAINTENANCE OF HOMEOSTASIS OF THE INTERNAL RESPIRATION REQUIRES THAT A FINE BALANCE BE MAINTAINED BETWEEN BLOOD FLOW AND ALVEOLAR VENTILATION IN THE LUNGS. WE HAVE SEEN THAT WHEN PRIMARY PATHOLOGICAL WEAKNESS OF THE HEART DEVELOPS, SO THAT THERE IS A DEFICIT IN DELIVERY OF OXYGEN TO THE CELLS, THERE IS A TENDENCY TO LOSE CO_2 BY HYPERVENTILATION. THIS MAY BE COMPENSATED BY ADAPTIVE CHANGES IN THE LUNGS, WHICH DIMINISH ALVEOLAR VENTILATION THUS PREVENTING THE DEVELOPMENT OF ACAPNIA

AND SHOCK. THE TRANSITION FROM CARDIAC SHOCK TO CONGESTIVE FAILURE MAY FREQUENTLY BE OBSERVED, AND THE PROGRESS OF THIS TRANSITION IS REFLECTED IN THE RISE IN CO_2 TENSION OF THE ARTERIAL BLOOD FROM THE HYPOCAPNIA OF SHOCK TO THE HYPERCAPNIA OF CONGESTIVE HEART FAILURE.

THE TYPES OF PATHOLOGICAL CONDITIONS WITHIN THE LUNGS WHICH LEAD TO PROGRESSIVE FAILURE TO MAINTAIN AN EFFECTIVE ALVEOLAR VENTILATION WILL NOW BE CONSIDERED. THEY ARE

- (1) EMPHYSEMA - OBSTRUCTIVE, POSTURAL, AND COMPENSATORY,
- (2) FIBROSIS, (3) PATCHY ATELECTASIS, (4) THICKENING OF ALVEOLAR WALLS, (5) PULMONARY ARTERIOLAR SPASM, SCLEROSIS, AND OCCLUSION, (6) BRONCHIAL SPASM, CONGESTION, AND EDEMA.

THE DEGREE OF EMPHYSEMA IS FREQUENTLY UNDERESTIMATED BOTH BY PHYSICAL EXAMINATION AND IN X-RAYS OF THE CHEST. ONE OF THE MOST IMPORTANT DIAGNOSTIC MEASURES IS THE DIRECT AUSCULTATION OF THE LUNGS WITH THE EAR AGAINST THE CHEST. ONLY IN THIS WAY WILL THE LOW INTENSITY OF THE VESICULAR MURMUR BE HEARD THROUGHOUT AND THE RELATIVE PROLONGATION OF THE EXPIRATORY MURMUR BE APPRECIATED. THE STETHOSCOPE IN SOME INSTANCES IS A HINDRANCE RATHER THAN A HELP.

FOR A QUANTITATIVE ESTIMATE OF EMPHYSEMA THE MEASUREMENT OF THE LUNG CAPACITY AND ITS SUBDIVISIONS IS IMPORTANT. AS A SINGLE INDEX THE RATIO RA/TC IS IMPORTANT. IN FIGURE 7

DATA FROM OUR LABORATORIES RELATES THIS RATIO TO THE O_2 SATURATION AND CO_2 CONTENT OF ARTERIAL BLOOD $\sqrt{20}$.

SLIDE 7

LEAVING ASIDE THE QUESTION AS TO WHETHER OR NOT THE METHODS WE THEN USED WERE SUFFICIENTLY PRECISE, THE RELATIONSHIP PRESENTED HERE REMAINS ESSENTIALLY TRUE. AS THE DEGREE OF EMPHYSEMA PROGRESSES ONE FINDS A PROGRESSIVE DECREASE IN ARTERIAL SATURATION WITH OXYGEN AND A PROGRESSIVE RISE IN CO_2 CONTENT OF THE ARTERIAL BLOOD. THE INCREASE IN CO_2 CONTENT IS COMPENSATED, AS A RULE, BY THE INCREASE IN ALKALINE RESERVE OF THE PLASMA BROUGHT ABOUT BY A SHIFT OF BASE TO THE PLASMA AND OF CHLORIDE TO THE TISSUES OR URINE. POLYCYTHEMIA MAY ASSIST IN THE INCREASED TRANSPORT OF CO_2 . DURING THE COMPENSATED PHASE THE PH OF THE BLOOD WILL NOT BE ALTERED. AS COMPENSATION BEGINS TO FAIL, THE TENSION OF CO_2 (PCO_2) BECOMES INCREASED. DUE TO THE FACT THAT CARBON DIOXIDE IS A NARCOTIC THE SENSITIVITY OF THE RESPIRATORY CENTER BECOMES DEPRESSED, SO THAT IT NO LONGER RESPONDS NORMALLY TO THE STIMULATION OF CO_2 . THIS EXPLAINS THE RESULTS OF THE SCOTT TEST $\sqrt{40}$.

SLIDE 8

IN THIS TEST THE NORMAL INDIVIDUAL WILL DOUBLE THE VENTILATION OF THE LUNGS WHEN 7 PER CENT CO_2 IS ADDED TO THE INSPIRED AIR, WHILE THE PATIENT WITH EMPHYSEMA INCREASES THE VENTILATION

SLIGHTLY OR NOT AT ALL, AND TOLERATES THE BREATHING OF PERCENTAGES OF CO_2 WHICH NORMALS CANNOT ENDURE. THIS TEST IS A MEASURE OF THE ADAPTATION TO A CHRONIC OR PERSISTENT HYPERCAPNIA.

ONCE THE RESPIRATORY CENTER HAS BECOME DEPRESSED BY CO_2 NARCOSIS, FURTHER DEPRESSION OF THE CENTER BY OTHER NARCOTICS, SUCH AS THE OPIUM ALKALOIDS MAY ^{PRODUCE} ~~PRECIPITATE~~ A RAPID AND CRITICAL RISE IN CARBON DIOXIDE TENSION AND ACIDOSIS, PRECIPITATING DECOMPENSATION OF THE PULMONARY FUNCTION AND SECONDARY FAILURE OF THE RIGHT VENTRICLE.

THE ADMINISTRATION OF OXYGEN MAY ALSO CONTRIBUTE TO THE SAME END, BY REMOVING THE ANOXIA WHICH IS ONE OF THE STIMULI STILL OPERATING DURING CO_2 NARCOSIS. STILL ANOTHER FACTOR WHICH MAY PRECIPITATE ^{it} IS THE INTERCURRENCE OF BRONCHIAL INFECTION OR A BRONCHOPNEUMONIA, OR A SUDDEN PNEUMOTHORAX WHICH TEND TO FURTHER DIMINUTION OF THE EFFECTIVE ALVEOLAR VENTILATION. THESE FACTORS WILL BE DISCUSSED IN MORE DETAIL LATER. THEY ARE MENTIONED AT THIS POINT IN ORDER TO EMPHASIZE THE IMPORTANCE OF RECOGNIZING THE MARGIN OF RESERVE OF VENTILATORY FUNCTION IN EMPHYSEMA.

IN ADDITION TO THE SCOTT TEST THERE ARE OTHER INDICATIONS OF IMPENDING FAILURE OF THE EMPHYSEMATOUS LUNG. THE VELOCITY OF BLOOD FLOW, AS MEASURED IN ARM TO TONGUE TIME, BECOMES MORE AND MORE ACCELERATED AS THE HYPERCAPNIA INCREASES. 14 ✓

IT IS NOT UNCOMMON TO FIND THE CIRCULATION TIME AS LOW AS 8 SECONDS IN EMPHYSEMA BEFORE THE RIGHT VENTRICLE FAILS. WHEN THIS FAILURE DOES OCCUR THE CIRCULATION TIME IS MORE PROLONGED, YET IT MAY STILL REMAIN WITHIN NORMAL LIMITS.

THE RESTING CARDIAC OUTPUT IS ALSO HIGH IN EMPHYSEMA BEFORE THE RIGHT VENTRICLE FAILS.

PULMONARY RESISTANCE TO BLOOD FLOW.

AS A FINAL STEP IN THE DIFFERENTIATION BETWEEN PRIMARY CARDIAC AND PRIMARY PULMONARY FAILURE, I WISH TO REVIEW RECENT ACQUISITIONS TO OUR KNOWLEDGE OF PULMONARY RESISTANCE TO BLOOD FLOW, AND THE RELATION TO IT OF THE RESPIRATORY GAS TENSIONS IN THE BLOOD.

YOU ARE, NO DOUBT, FAMILIAR WITH THE DISCOVERY OF VON EULER AND LILJESTRAND¹², THAT ANOXIA RAISES THE PRESSURE IN THE PULMONARY ARTERY OF THE CAT. THIS FACT HAS BEEN CONFIRMED IN MAN BY MOTLEY, *et al*³⁴, AND IN OTHER LABORATORIES INCLUDING OUR OWN.

DIRKEN AND HEEMSTRA⁸ ⁹✓ CONFIRMED THE RISE IN PULMONARY PRESSURE AND ATTRIBUTED IT TO THE FORMATION OF HISTAMINE IN THE ANOXIC LUNG. IF ONE LUNG WERE VENTILATED WITH A LOW OXYGEN MIXTURE - PULMONARY VASOCONSTRICTION OCCURRED ON THAT SIDE, THUS DIVERTING BLOOD TO THE NORMALLY OXYGENATED LUNG .

VASOMOTOR NERVES APPEAR TO PLAY LITTLE PART IN THIS EFFECT.

CLAIMS HAVE BEEN MADE THAT THE HISTAMINE CONTENT OF THE ARTERIAL BLOOD IS ELEVATED IN ANOXIA, / AND, MORE RECENTLY THAT THE HISTAMINE CONTENT OF ARTERIAL BLOOD IS HIGHER THAN OF THE VENOUS.

IN OUR OWN "CHEST LABORATORY" BLOOD HISTAMINE DETERMINATIONS IN ANOXIC PATIENTS HAVE BEEN MADE, USING A METHOD DESCRIBED BY DR. HELEN GRAHAM /. THE RESULTS OF THESE DETERMINATIONS HAVE NOT BEEN PUBLISHED, BECAUSE SO FAR THEY HAVE NOT BEEN AT ALL CONSISTENT. IN OUR VASCULAR LABORATORY, DURWOOD SMITH AND MARGARET CUSHMAN HAVE MADE UNPUBLISHED DETERMINATIONS OF HISTAMINE OF THE BLOOD AND ALSO OF THE LUNGS OF ANOXIC PIGS. THE AMOUNT OF HISTAMINE IN THE LUNG APPEARS TO BE GREATLY INCREASED. THE VARIABLE QUANTITIES OF HISTAMINE IN THE BLOOD MAY BE EXPLAINED BY VARIABLE RETENTION OR DESTRUCTION OF HISTAMINE IN THE LUNG. THIS WHOLE QUESTION OF HISTAMINE IN ANOXIA NEEDS FURTHER CLARIFICATION, BUT THE VIEWS OF DIRKEN AND HEEMSTRA ARE PROBABLY CORRECT. THE KNOWN ACTIONS OF HISTAMINE ARE SUCH AS WOULD CONSTRICT THE PULMONARY VESSELS AND DILATE THE ALVEOLAR CAPILLARIES.

DURING THE PAST YEAR WE HAVE MADE ESPECIAL EFFORTS TO DETERMINE THE EFFECT OF CARBON DIOXIDE ON THE PULMONARY RESISTANCE AND BLOOD FLOW. THESE RESULTS ARE AT PRESENT BEING PREPARED FOR PUBLICATION. IT CAN BE SAID HERE THAT 3.6 PER CENT

CO₂ AND 6 PER CENT CO₂ WITH 21 PER CENT OXYGEN WERE GIVEN TO NORMALS AND PATIENTS WITH EMPHYSEMA. IN THE NORMALS VENTILATION AND BLOOD FLOW INCREASED IN RESPONSE TO INSPIRED CO₂, WITH VERY LITTLE RISE IN PULMONARY ARTERIAL PRESSURE (+9 PER CENT). PATIENTS WITH SEVERE EMPHYSEMA SHOWED LITTLE OR NO INCREASE IN VENTILATION AND A MUCH LARGER RISE IN PULMONARY ARTERIAL PRESSURE (28-106 PER CENT). I SHALL TAKE THE LIBERTY OF SHOWING A TABULATION OF THE CORRELATIONS BETWEEN THE VARIOUS DETERMINANTS FROM THE UNPUBLISHED PAPER OF MY ASSOCIATES, DOCTORS LOVEJOY, YU, JOOS AND NYE.

SLIDE 9

THESE DATA WERE ALL OBTAINED IN A STUDY OF PATIENTS WITH CHRONIC PULMONARY DISEASE.

CORRELATION BETWEEN VARIOUS DETERMINANTS IN 18 PATIENTS
WITH PULMONARY EMPHYSEMA

| | MEAN PUL. ART. PRESS. | MEAN PUL. "CAP" PRESS. | TOTAL PUL. RESISTANCE | PULMONARY ARTERIOULAR RESISTANCE |
|---|--------------------------|---------------------------|--------------------------|--|
| ARTERIAL BLOOD PCO ₂ | +0.693** | +0.284 | +0.742** | +0.651** |
| MIXED VENOUS BLOOD PCO ₂ | +0.613** | +0.200 | +0.704** | +0.582* |
| RA/TC x 100 | +0.624** | +0.081 | +0.544* | +0.420 |
| ARTERIAL BLOOD O ₂ SATURATION | -0.490* | -0.002 | -0.318 | -0.312 |
| CARDIAC INDEX | -0.396 | +0.208 | -0.518* | -0.452 |
| ARTERIAL BLOOD PO ₂ | -0.426 | +0.093 | -0.085 | -0.272 |
| MIXED VENOUS BLOOD PO ₂ | -0.050 | -0.246 | -0.080 | -0.001 |
| RIGHT AURICULAR PRESSURE | +0.292 | +0.643** | +0.279 | -0.114 |

** HIGHLY SIGNIFICANT

* SIGNIFICANT

YOU WILL NOTE THE VERY HIGH AND SIGNIFICANT POSITIVE CORRELATION OF THE ARTERIAL PCO_2 WITH PULMONARY RESISTANCE. THE MIXED VENOUS PCO_2 IS ALMOST AS HIGHLY CORRELATED AND THE RATIO RA/TC ALSO QUITE HIGH AND SIGNIFICANT. RELATIVE TO CARBON DIOXIDE - THE OXYGEN TENSIONS AND BLOOD FLOW SHOW LESS SIGNIFICANT AND NEGATIVE CORRELATIONS WITH PULMONARY RESISTANCE.

ALMOST UNIFORMLY RECENT STUDIES OF THE HEART FAILURE PROBLEM HAVE CENTERED ON THE INADEQUATE DELIVERY TO THE CELLS OF THE BODY ENOUGH OXYGEN TO SATISFY THE DEMANDS OF THE METABOLISM. THIS VIEW WAS WELL DEVELOPED BY J. M. LITTLE²⁶ ✓. ALSO COURNAND IN HIS HAMBURGER LECTURE⁶ ✓ ON THE PULMONARY CIRCULATION GIVES MAJOR ATTENTION TO THE FACTOR OF OXYGEN LACK AND LITTLE OR NO DISCUSSION OF THE CO_2 FACTOR. MORE RECENTLY WESTCOTT AND CO-WORKERS⁴³ ✓ IN A STUDY OF ANOXIA AND PULMONARY VASCULAR RESISTANCE PAY LITTLE ATTENTION TO THE CO_2 FACTOR, EXCEPT THAT THEY OBSERVED THE EFFECT OF GIVING 5 PER CENT CO_2 IN INSPIRED AIR TO FIVE NORMAL SUBJECTS, BUT NO PATIENTS WITH EMPHYSEMA WERE SO OBSERVED. WESTCOTT FOUND, AS WE HAVE, THAT IN NORMALS THE EFFECTS OF INSPIRED CO_2 ON PULMONARY RESISTANCE WERE VARIABLE AND SLIGHT. OUR RESULTS IN EMPHYSEMATOUS PATIENTS, WHEN THEY ARE PUBLISHED, WILL CONVINCE YOU I AM SURE, THAT THE CO_2 FACTOR IS AT LEAST EQUAL TO IF NOT GREATER THAN THE ANOXIA FACTOR IN CHRONIC PULMONARY DISEASE.

THE GREAT FALLACY IN THIS EXCLUSIVE CONSIDERATION OF ANOXIA LIES IN ~~THE FACT~~ THAT IT DOES NOT ACCOUNT FOR THE FACT THAT ANOXIA, UNDER ONE SET OF CIRCUMSTANCES, LEADS TO SHOCK, WHILE IN OTHERS IT RESULTS IN CONGESTIVE FAILURE. ²⁴✓ ²⁸✓

IN OUR LABORATORY AND CLINIC WE HAVE BECOME CONVINCED OF THE FUNDAMENTAL RIGHTNESS OF THE VIEWS OF YANDELL HENDERSON ¹⁶✓ ¹⁷✓, WHO OBSERVED THAT ANOXIA LED TO HYPERVENTILATION WITH ACAPNIA AND SHOCK IN SUCH CONDITIONS AS LOBAR PNEUMONIA, AND CARBON MONOXIDE POISONING. THE SAME THING IS TRUE IN MOUNTAIN SICKNESS. ON THE OTHER HAND BRONCHOPNEUMONIAS, WITH THEIR COMBINATION OF EMPHYSEMA AND ATELECTASIS CHARACTERISTICALLY LEAD TO CONGESTIVE FAILURE. THE DIFFERENCE IN THE TWO SITUATIONS IS TO BE EXPLAINED ON TWO COUNTS. FIRST, IT DEPENDS ON WHAT HAPPENS TO THE VENTILATION. IF ANOXIC HYPERVENTILATION IS NOT CHECKED, ACAPNIA AND SHOCK ARE INEVITABLE. IF ADAPTIVE CHANGES IN THE LUNG OCCUR, SUCH AS THE LOCAL DEVELOPMENT OF HISTAMINE EFFECTS ON THE BRONCHI, THE RA/TC RATIO WILL INCREASE, AND PULMONARY CONSTRICTION WILL OCCUR, AND THE LOSS OF CO₂ WILL BE CHECKED. IN LOBAR PNEUMONIA THE LOSS OF CO₂ IS NOT ALWAYS CHECKED, BECAUSE ALTHOUGH BLOOD FLOW IS SHIFTED TO UNINVOLVED LOBES, THE RAPID SHALLOW BREATHING CONTINUES TO HYPERVENTILATE THEM. IN BRONCHOPNEUMONIAS (AND I REFER TO CATARRHAL NOT INTERSTITIAL PROCESSES) THE VENTILATION IS IMPAIRED AND CO₂ TENDS TO ACCUMULATE BEYOND NORMAL LEVELS. SECOND, THE EFFECTS OF CARBON DIOXIDE ON

THE PERIPHERAL CIRCULATION HAS TO BE TAKEN INTO ACCOUNT.

CARBON DIOXIDE IS A POWERFUL PHARMACODYNAMIC AGENT. IN GENERAL HYPOCAPNIA TENDS TO SLOW DOWN THE RATE AND VOLUME OF BLOOD FLOW. THIS IS WELL SHOWN IN ITS EFFECTS UPON THE CEREBRAL BLOOD FLOW. (KETY, SCHMIDT, ET AL). EXCESS OF CO₂ TENDS TOWARD VASODILATION ^{and the opening of shunts,} TOWARD ACCELERATION OF THE RATE AND VOLUME OF BLOOD FLOW UP TO THE POINT WHERE THE RIGHT VENTRICLE AND PULMONARY CIRCUIT CAN NO LONGER ACCOMODATE THE FLOOD OF VENOUS BLOOD POURED INTO IT.

WE SHOULD NOT CLOSE THIS TALK WITHOUT SOME ATTENTION TO THE CEREBRAL CIRCULATION IN PULMONARY FAILURE. ATTENTION HAS BEEN CALLED TO THE FACT THAT SOME CYANOTIC PATIENTS WITH EMPHYSEMA MAY LAPSE INTO COMA WHEN OXYGEN IS GIVEN, AND RECOVER CONSCIOUSNESS WHEN IT IS WITHDRAWN. THE CEREBROSPINAL PRESSURE HAS BEEN SHOWN TO RISE AS THE CYANOSIS CLEARS, AND TO FALL AS IT RETURNS. AN ILLUSTRATIVE EXAMPLE OF THIS IS SHOWN IN

Table
FIGURE 10. 2. ✓

Insert table 2. (^{SLIDE 10}_{p. 23-as})

SLIDE 11

IN FIGURE 11 IS SHOWN A MORE EXTENDED OBSERVATION OF SUCH AN EVENT IN A WOMAN WITH EMPHYSEMA AND BRONCHITIS, WHO WAS PUT ALMOST IN EXTREMIS BY THE ADMINISTRATION OF OXYGEN AND

TABLE 2 ²⁹

EFFECT OF ADMINISTERING PURE OXYGEN BY PRESSURE
MASK ON THE BLOOD GASES AND pH OF BLOOD OF A
PATIENT WITH OBSTRUCTIVE EMPHYSEMA, WITH
CONCOMITANT CHANGES IN INTRATHECAL
PRESSURE

Oxygen capacity23.1 vol. per cent
Hematocrit51 per cent

| | BEFORE OXYGEN MASK | AFTER 15 MINUTES OF OXYGEN |
|------------------------------------|-----------------------|-------------------------------|
| Oxygen content | 16.15 vol. per cent | 22.72 vol. per cent |
| Oxygen saturation | 69.9 vol. per cent | 98.4 vol. per cent |
| pCO ₂ mm. of mercury | 71.9 | 87.5 |
| pH | 7.3 | 7.18 |

SPINAL FLUID PRESSURE

| | | |
|---------------------|-----|-----|
| mm.H ₂ O | 160 | 260 |
|---------------------|-----|-----|

RESCUED BY ARTIFICIAL HYPERVENTILATION IN A DRINKER RESPIRATOR, FOLLOWING A PROCEDURE WHICH WHITTENBERGER / HAD SUCCESSFULLY EMPLOYED. THIS CASE HAS BEEN REPORTED (McCANN, LOVEJOY AND YU³⁰✓).

UP TO THIS POINT I HAVE DRAWN IN SHARPLY CONTRASTING BLACK AND WHITE A PICTURE OF OUR CONCEPT OF PRIMARY HEART FAILURE (LEFT VENTRICULAR) AND THAT OF RIGHT HEART FAILURE SECONDARY TO PULMONARY EMPHYSEMA. A BRIEF AND SKETCHY RECAPITULATION OF THEIR GENESIS IS AS FOLLOWS:

ANOXIA

LEFT HEART FAILURE

1. HYPERVENTILATION WITH CO_2 LOSS.
2. HISTAMINE IN LUNG.
Response to anoxia.
3. PULMONARY VASO CONSTRICTION AND INCREASE R.A.
DECREASE VC.
4. EUCAPNIA OR MODERATE HYPERCAPNIA WITH RISING RA/TC.
5. SLOWING OF CIRCULATION RATE
AND DIMINISHING CARDIAC OUTPUT.
6. SECONDARY FAILURE OF RIGHT VENTRICLE.

RIGHT HEART FAILURE

1. CO_2 ACCUMULATES DUE TO INCREASE RA/TC.
2. COMPENSATION - INCREASE NA AND DECREASE CL IN PLASMA.
3. MARKED INCREASE PCO_2 + NARCOSIS OF RESPIRATORY CENTER.
4. DECREASE EFFECTIVE VENTILATION.
5. INCREASE IN VENOUS INPUT. INCREASE IN PULMONARY RESISTANCE.
6. FAILURE RIGHT VENTRICLE

IN TRACING THE COURSE OF EVENTS WE MUST CONSIDER THAT THE TENSIONS OF O_2 AND CO_2 MAY HAVE DIFFERENT EFFECTS AT DIFFERENT POINTS

IN THE CARDIORESPIRATORY MECHANISM. FOR INSTANCE:

LOW ARTERIAL PO_2 MAY DEPRESS THE RESPIRATORY CENTER A LITTLE - BUT THIS IS OFFSET BY THE STIMULATION OF PERIPHERAL CHEMO-RECEPTORS. NET RESULT HYPERVENTILATION. AT THE SAME TIME THE FORMATION OF HISTAMINE IN THE LUNG ~~PLUS~~ ^S ALTERING THE PULMONARY RESISTANCE AND ALVEOLAR VENTILATION AND COUNTERACTS CO_2 LOSS THROUGH HYPERVENTILATION.

LOW PCO_2 ⁱⁿ ARTERIAL BLOOD ^{is} ASSOCIATED GENERALLY WITH A DIMINUTION OF RATE AND VOLUME OF BLOOD FLOW, HIGH PCO_2 WITH INCREASED RATE AND VOLUME, AT THE SAME TIME AUGMENTING THE PULMONARY RESISTANCE.

IN DRAWING THE CONTRASTING PICTURE OF PRIMARY HEART FAILURE AND PULMONARY HEART FAILURE WE HAVE BEEN OBLIGED TO OMIT DETAILS OF CERTAIN CASES OF PULMONARY FIBROSIS IN WHICH EMPHYSEMA IS VERY SLIGHT OR NON-APPARENT; DYSPNEA MAY BE MARKED AND EFFORT GREATLY LIMITED. I REFER TO CASES IN WHICH THE RESTING LUNG VOLUME MAY BE DECREASED AND THE RATIO OF RA/TC NOT GREATLY ALTERED, ALTHOUGH X-RAYS MAY REVEAL EVIDENCE OF FIBROTIC CHANGE, OR EVEN THOSE SUGGESTIVE OF AN IRREGULAR EMPHYSEMA. THESE PATIENTS MAY SHOW NO HYPERCAPNIA; THEY MAY SHOW NO ELEVATION OF RESTING PULMONARY RESISTANCE AT REST. THE SYSTEMIC BLOOD PRESSURE MAY BE QUITE VARIABLE - LOW OR NORMAL IN RANGE. ANOXIA AS EXPRESSED IN ARTERIAL SATURATION MAY BE VARIABLE FROM

ONE OBSERVATION TO ANOTHER. DYSPNEA MAY BE PRONOUNCED AT REST AND EFFORT GREATLY LIMITED. RESTING RECORDS OF THE RESPIRATION MAY BE VERY IRREGULAR, OR THERE MAY BE UNDULATORY CHANGES IN THE MID-CAPACITY LEVELS, AS THOUGH THERE WERE EXAGGERATED PERISTALSIS OR GREAT VARIATIONS IN TONUS OF THE BRONCHIAL MUSCULATURE. MANY PERMUTATIONS AND COMBINATIONS OF THESE FINDINGS WILL BE OBSERVED, WHICH RENDER THEM MOST DIFFICULT TO INTERPRET. THESE CASES DO NOT SHOW THE PICTURE OF CONGESTIVE FAILURE.

IT IS MY BELIEF THAT THESE VARIATIONS ARE DUE TO A WIDE RANGE OF CAUSES. ONE MAY, ON FURTHER INVESTIGATION, FIND AN UNSUSPECTED RENAL ACIDOSIS, FOR INSTANCE, OR LATENT CORONARY DISEASE. THEY MAY REVEAL EVIDENCE OF CEREBRAL ARTERIOSCLEROSIS. THEY MAY HAVE POT BELLIES WITH THIN, WEAK MUSCLES AND VARICOSE VEINS. ONE SUSPECTS THAT THE SENSITIVITY OF THE CHEMORECEPTORS BOTH IN AND OUTSIDE THE CHEST MAY BE ENHANCED IN SOME OF THESE PEOPLE. ONE MAY SUSPECT THAT ANOXIA MAY NOT INDUCE HISTAMINE FORMATION UNIFORMLY IN ALL PERSONS. CERTAINLY INDIVIDUALS VARY IN THEIR SUSCEPTIBILITY TO ALLERGIC DISORDERS. NOT INFREQUENTLY ONE OBSERVES MARKED ANXIETY, WHICH COULD PLAY A ROLE IN THE HYPERVENTILATION OBSERVED.

SO - WHILE ONE MAY PAINT A CLINICAL PICTURE - A TEXTBOOK PICTURE - OF HEART FAILURE FOLLOWING MARKED EMPHYSEMA, WITH

ANOXIA AND HYPERCAPNIA, HE IS FACED WITH A MULTITUDE OF VARIANTS FROM THIS PICTURE. THE INTERPRETATION OF THE CURRENTLY USED TESTS FOR STUDY OF THE LUNGS AND PULMONARY CIRCULATION REQUIRES ALL THE RESOURCES NOW AVAILABLE TO THE MODERN INTERNIST, AND EVEN THEN THE CONCLUSIONS REACHED CAN BE ONLY TENTATIVE. AS USUAL THE MOST DIFFICULT FACTORS TO EVALUATE ARE THE PSYCHOLOGICAL ONES AND THE NEURO-PHYSIOLOGICAL.

IN SUMMARY PERMIT ME TO QUOTE WILLIAM HARVEY: -

"THIS IS EVIDENCE OF TWO KINDS OF DEATH, FAILURE FROM A LACK, AND SUFFOCATION FROM AN EXCESS. IN THESE EXAMPLES OF BOTH, ONE MAY FIND PROOF BEFORE HIS EYES OF THE TRUTH SPOKEN ABOUT THE HEART."

"FAILURE FROM A LACK" IS SHOCK. THE LACK IS OF BOTH OXYGEN AND CO_2 , ANOXIA AND ACAPNIA, WITH RESULTANT SLOWING OF THE CIRCULATION IN RATE AND VOLUME. THIS SITUATION MAY OCCUR FROM ACUTE ISCHEMIC OR TOXIC INJURY TO THE LEFT VENTRICLE. IT MAY ALSO OCCUR IN FIBROSES OF THE LUNG WHICH ARE NOT ACCOMPANIED BY EMPHYSEMA, AS WELL AS IN CO POISONING AND LOBAR PNEUMONIA.

"SUFFOCATION FROM AN EXCESS" IS CONGESTIVE FAILURE OF THE CIRCULATION. WHEN THIS IS DUE TO PRIMARY DISEASE OF THE HEART, THE RATE AND VOLUME OF CIRCULATION ARE DECREASED BY THE "CHECK-VALVE" ACTION OF THE LUNG, DUE TO LOCAL ACTION

OF HISTAMINE, WHICH DIMINISHES ALVEOLAR VENTILATION AND INCREASES ARTERIAL RESISTENCE AND COMPENSATES FOR THE TENDENCY OF ANOXIC HYPERVENTILATION TO PRODUCE ACAPNIA, *by narrowing of the bronchi.*

WHEN IT IS DUE TO PRIMARY PULMONARY LESIONS ASSOCIATED WITH EMPHYSEMA, THE VENOPRESSOR EFFECTS OF HYPERCAPNIA POUR A FLOOD OF VENOUS BLOOD INTO THE RIGHT HEART, WHILE AT THE SAME TIME THE "CHECK-VALVE" MECHANISMS OF THE LUNG INCREASE THE RESISTANCE TO TRANSFER OF BLOOD TO THE LEFT VENTRICLE, SO THAT THE RIGHT HEART IS OVERWHELMED. WHEN SEVERE ANOXIA OCCURS, THE EVENTS, WHICH DETERMINE WHICH FORM OF DEATH WILL ENSUE, TAKE PLACE WITHIN THE LUNG.

BIBLIOGRAPHY

1. ALTSCHULE, M.D., THE PATHOLOGICAL PHYSIOLOGY OF CHRONIC CARDIAC DECOMPENSATION, MEDICINE, 17:75-154, 1938.
2. BOUTOURLINE-YOUNG, H.J., AND WHITTENBERGER, J.L., THE USE OF ARTIFICIAL RESPIRATION IN PULMONARY EMPHYSEMA ACCOMPANIED BY HIGH CARBON DIOXIDE LEVELS, J. CLIN. INVESTIGATION, 30:838, AUG. 1951.
3. BURKHARDT, W.L., FLICKINGER, D., AND ADLER, H.F., BLOOD HISTAMINE IN HYPOXIA, FED. PROC., 8:19, MARCH 1949.
4. COMROE, J.H., JR., BAHNSON, E.R., AND COATES, E.O., MENTAL CHANGES OCCURRING IN CHRONICALLY ANOXEMIC PATIENTS DURING OXYGEN THERAPY, J.A.M.A., 143: 1044, 1950.
5. Cournand, A. and Ranges, H.A., CATHETERIZATION OF THE RIGHT AURICLE IN MAN, PROC. SOC. EXPER. BIOL. MED., 46:462, 1941.
6. Cournand, A., SOME ASPECTS OF THE PULMONARY CIRCULATION IN NORMAL MAN AND IN CHRONIC CARDIOPULMONARY DISEASES, CIRCULATION, 2:641, 1950.
7. DILL, D.B., LIFE, HEAT AND ALTITUDE, HARVARD UNIV. PRESS, CAMBRIDGE, 1938.
8. DIRKEN, M.R.J., AND HEEMSTRA, H., ALVEOLAR OXYGEN TENSION AND LUNG CIRCULATION, Q.J. EXPER. PHYSIOL. 34:193-211, 1948.
9. DIRKEN, M.R.J., AND HEEMSTRA, H., THE ADAPTATION OF THE LUNG CIRCULATION TO THE VENTILATION, Q. J. EXPER. PHYSIOL., 34:213-226, 1948.
10. DIRKEN, M.M., AND HEEMSTRA, H., AGENTS ACTING ON THE LUNG CIRCULATION, Q.J. EXPER. PHYSIOL., 34:227-242, 1948.
11. DONALD, K., NEUROLOGICAL EFFECTS OF OXYGEN, LANCET, 2:1056, Nov. 1949.
12. v. EULER, V.S., AND LILJESTRAND, G., OBSERVATIONS ON THE PULMONARY ARTERIAL PRESSURE IN THE CAT, ACTA. PHYSIOL. SCAND., 12:301, 1946.

13. FERGUSON, F.C., KOBILAK, R.E., AND DIETRICH, J.E., VARICES OF THE BRONCHIAL VEINS AS A SOURCE OF HEMOPTYSIS IN MITRAL STENOSIS, AM. HEART J., 28:445-456, 1944.
14. GILLANDERS, A.D., CIRCULATORY DYNAMICS IN EMPHYSEMA, Q. J. MED. N.S., 18:263-273, 1949.
15. HARVEY, WILLIAM, ANATOMICAL STUDIES OF THE MOTION OF THE HEART AND BLOOD. "DE MOTU CORDIS" - 1628, TRANSLATION BY CHAUNCEY D. LEAKE, CHAP. X, P. 84, CHARLES C. THOMAS, SPRINGFIELD, 1931.
16. HENDERSON, Y., TONUS AND THE VENOPRESSOR MECHANISM. THE CLINICAL PHYSIOLOGY OF A MAJOR MODE OF DEATH, MEDICINE, 22: 222-350, 1943.
17. HENDERSON, Y., ADVENTURES IN RESPIRATION, WILLIAMS AND WILKINS, BALTIMORE, 1938.
18. HEYMANS, C., INTRODUCTION TO THE REGULATION OF BLOOD PRESSURE AND HEART RATE, AMERICAN LECTURES IN PHYSIOLOGY, ED. R.F. PITTS, C.C. THOMAS PUBL., SPRINGFIELD, ILL.
19. HURTADO, A., KALTREIDER, N.L., AND McCANN, W.S., RESPIRATORY ADAPTATION TO ANOXEMIA, AM. J. PHYSIOL., 109:626-637, 1934.
20. HURTADO, A., KALTREIDER, N.L., AND McCANN, W.S., STUDIES OF THE TOTAL PULMONARY CAPACITY AND ITS SUBDIVISIONS. IX. RELATIONSHIP TO THE OXYGEN SATURATION AND THE CARBON DIOXIDE CONTENT OF THE ARTERIAL BLOOD, J. CLIN. INVESTIGATION, 14:94-105, 1935.
21. KAUMP, D.H. AND DRY, T.J., PULMONARY ARTERIOLAR SCLEROSIS, ARCH. INT. MED., 61, 1-18, 1938.
22. LAUSON, H.D., BLOOMFIELD, R.A., AND COURNAND, A., THE INFLUENCE OF THE CIRCULATION ON THE RESPIRATION OF MAN, AM. J. MED., 4:193-200, 1948.
23. LEVY, M.N., AND BERNE, R.M., PRODUCTION OF ACUTE EXPERIMENTAL CIRCULATORY FAILURE BY GRADED PULMONARY ARTERY CONSTRICTION # (17358), PROC. SOC. EXPER. BIOL. MED., 72: 147-153, 1949.
24. LEWIS, R.B., AND HAYMAKER, W., HIGH ALTITUDE HYPOXIA. OBSERVATIONS AT AUTOPSY IN 75 CASES, AND ANALYSIS OF CAUSES OF HYPOXIA, J. AVIATION MED., 19:306-336, 1948.

26. LIEBOW, A.A., HALES, M.R., AND LINDSKOG, G.E., ENLARGEMENT OF THE BRONCHIAL ARTERIES AND THEIR ANASTOMOSES WITH THE PULMONARY ARTERIES IN BRONCHIECTASIS, *AM. J. PATH.*, 25: 211-232, 1949.
26. LITTLE, J.M., A UNIFIED CONCEPT OF CARDIAC FAILURE, *AM. J. MED.*, 7:207-215, 1949.
27. McCANN, W.S., BRUCE, R.A., LOVEJOY, F.W., JR., YU, P.N.G., PEARSON, R., EMERSON, E.B., ENGEL, G., AND KELLY, J.J., TUSSIVE SYNCOPE: OBSERVATIONS ON THE DISEASE FORMERLY CALLED "LARYNGEAL EPILEPSY", *ARCH. INT. MED.*, 84:845-846, 1949.
28. McCANN, W.S., SOME NEGLECTED ASPECTS OF CARDIOLOGY. CIRCULATORY FUNCTION OF THE RESPIRATION, *AM. J. MED.* 8:62-70, JAN. 1950.
29. McCANN, W.S., THE EFFECTS OF EMPHYSEMA OF THE LUNGS ON THE CEREBRAL AND CORONARY CIRCULATION, *POSTGRADUATE MED.*, VOL. 9, No. 3, 225-230, MARCH 1951.
30. McCANN, W.S., LOVEJOY, F.W., JR., AND YU, P.N.G., THE FAILING LUNG, *N.Y. STATE J. OF MED.*, 52:1983, AUG. 15, 1952.
31. McMICHAEL, J., HEART FAILURE OF PULMONARY ORIGIN, *EDIN. MED. J.*, 55:65-77, 1948.
32. MILLER, A.T., ACCLIMATIZATION TO CARBON DIOXIDE, *AM. J. PHYSIOL.*, 129:524, JUNE 1940.
33. MILLER, W.S., THE LUNG, CHARLES C. THOMAS, SPRINGFIELD, ILL., 1937. (P. 63)
34. MOTLEY, H.L., COURNAND, A., WERKE, L., HIMMELSTEIN, A., AND DRESDALE, D., EFFECT OF SHORT PERIODS OF INDUCED ANOXIA UPON THE PULMONARY ARTERIAL PRESSURE IN MAN, *AM. J. PHYSIOL.*, 150:315, 1947.
35. PARKER, R. L., PULMONARY EMPHYSEMA, *ANN. INT. MED.*, 14:795-809, 1940.
36. PI-SUÑER, A., THE REGULATION OF RESPIRATORY MOVEMENTS BY PERIPHERAL CHEMORECEPTORS, *PHYSIOL. REV.*, 27:1-38, JAN. 1947.
37. RAKSHIT, P., COMMUNICATING BLOOD VESSELS BETWEEN BRONCHIAL AND PULMONARY CIRCULATIONS IN THE GUINEA PIG AND RAT, *Q. J. PHYSIOL.*, 35:47-53, 1949.

38. ROUVIERE, H., ANATOMY OF THE HUMAN LYMPHATIC SYSTEM, EDWARDS BROS., ANN ARBOR, 1938.
39. SCHLAEPFER, K., LIGATION OF THE PULMONARY ARTERY OF ONE LUNG WITH OR WITHOUT SECTION OF THE PHRENIC NERVE, ARCH. SURG., 9:25-94, 1924.
40. SCOTT, R.W., OBSERVATIONS ON THE PATHOLOGICAL PHYSIOLOGY OF CHRONIC PULMONARY EMPHYSEMA, ARCH. INT. MED., 26:544, 1920.
41. SEEVERS, M.H., THE NARCOTIC PROPERTIES OF CARBON DIOXIDE, N.Y.S. J. MED., 44:597, 1944.
42. VIRCHOW, R., UEBER DIE STANDPUNKT IN WISSENSCHAFTLICHE MEDICINE, ARCH. F. PATH. ANAT. U. PHYSIOL., 1:19, 1847. IBID., 3:456, 1851.
43. WESTCOTT, R.N., FOWLER, N.O., SCOTT, R.C., HAUENSTEIN, V.D., AND MCGUIRE, J., ANOXIA AND HUMAN PULMONARY VASCULAR RESISTANCE, J. CLIN. INVEST., 30:957-970, 1951.
44. WOOD, D.A., AND MILLER, M., THE ROLE OF THE DUAL PULMONARY CIRCULATION IN VARIOUS PATHOLOGICAL CONDITIONS OF THE LUNGS, J. THORAC. SURG., 7:649-670, 1937-38.

BY DOCTOR WARING:

Doctor McCann and Members of the Symposium, Doctor McCann has, as always, presented factual matter as well as philosophical comments with such a happy facility that I envy him. I'm reminded of the story which I hope our British friends here will not find incorrect, about Sir Joseph

Discussion

James J. Waring, M.D.

Dr. McCann has covered.....

.....(Dr. Waring's paper on file).....

.....did not increase.

Thurs AM.

Rec'd at San. Hall

1/26/53

WRS

CARDIO-CIRCULATORY ASPECTS OF THE PNEUMONOCONIOSES

by

James J. Waring, M.D., M.A.C.P.

Dr. McCann has covered his assignment so beautifully that I am reminded of the story of the visit of Sir Joseph Lister to a formal occasion of a great English university where he made a splendid address. Following Sir Lister's address there was a demand from the audience for the local Professor of Surgery to say something. As it happened the local professor was extremely modest and it required much and very flattering insistence from the audience to make him step forward on the platform. When the audience became still he looked them over and spoke as follows: "When the English Nightingale sings all other birds should be still." He then returned to his seat. Perhaps following this example the Rocky Mountain Magpie should be quiet since the Rochester Nightingale has sung so beautifully.

As always, Dr. McCann has given a very thoughtful discussion of the varied problems facing physiologists and clinicians in dealing with the cardio-circulatory ^{aspects of the} pneumonoconioses. Within certain limitations he sings the praise of CO₂.

In the past we have indeed erred too much in concentrating attention on ventilation to the neglect of the pulmonary circulation. We have been too intent on the obvious importance of getting oxygen into the lungs and too little aware of the importance of getting carbon dioxide and nitrogen out of the lungs. It is not paradoxical to say that we have been perhaps too much interested in "heart failure" to the neglect of "lung failure." In "lung failure" both pulmonary and systemic circulation are involved and McCann is quite right when he says, "The inter-relationship of the functions of ventilation and circulation are so intimate that one cannot think of one

without the other."

As to the significance of emphysema in the pneumoconioses, the difficulty in its recognition and its physiological significance, I am in agreement. McCann says emphysema is the chief cause in the genesis of right heart failure.

In its advanced, diffuse form, emphysema is distinguished by its "absences":

1. Absent or diminished cardiac and hepatic dullness
2. Absent or diminished diaphragmatic movement
3. Absent or diminished breath sounds
4. Absent or diminished broncho-vascular markings by fluoroscopy
5. Absent or diminished broncho-vascular markings in the roentgenogram
6. Absent filling of the finer bronchi with lipiodol in the bronchogram
7. Absent retention of lipiodol after the bronchogram

In physical diagnosis, absences are always less conspicuous than presences.

Some years ago R. W. Scott (Arch. of Int. Med. 26:544, 1920) noted the remarkable tolerance of the large lunged emphysematous patient for breathing for short periods of time, say only ten to fifteen minutes, high concentrations (8 - 10%) of carbon dioxide. Added carbon dioxide to the inhaled air, with oxygen, did not materially stimulate the ventilation of these patients. However, Scott noted the appearance of acute distress at certain higher critical concentrations of carbon dioxide in these patients. One patient, comfortable on breathing 9.28% CO₂, was in great distress after

breathing 11.44% CO₂. At this concentration his minute volume was only 14 L/m. In other words, with a potent stimulus to respiration, he had a factor of safety amounting to about 7 L/m as compared with about 50 L/m for normal persons under the same conditions. This indicates a marked limitation in pulmonary reserve.

One of Scott's charts showed that when the normal subject breathed 8% CO₂ the tidal air was increased 300%; but in the emphysematous patient only 25%. Note the significance of the prolonged expiration phase of the respiratory cycle. Prolonged emptying time explains the reduction of M.B.C. Scott concludes, "The level at which carbon dioxide equilibrium is established is definitely higher in emphysema than in the normal."

Through the generosity of Dr. L. L. Anderson of the Cardio-Pulmonary Division at the University of Colorado Medical Center, I can give you interesting but incomplete reports on a group of patients with emphysema due to various causes, namely asthmatic bronchitis and silicosis. I have divided these 19 patients into two groups, 12 averaged 60 years of age; 7 averaged 45 years of age. The older group had a residual air of 61.2%, the younger group a residual air of 41%. The older group had an M.B.C. of 33.5 liters, the younger group 73.1 liters. The older group had an average arterial oxygen saturation at rest of 71% and the younger group an average arterial oxygen saturation at rest of 91%. For seven patients over 60 years of age, the M.B.C. averaged 26.4 liters, the R.A. 61.4% and the arterial oxygen saturation 75%. The average pulmonary arterial pressure for the 12 patients over 60 years of age was much higher than for the seven persons under 45 years of age. Anderson has studied some 50 emphysematous patients, half with severe emphysema and the other half with moderate emphysema. None were in heart failure at the time of study. All had either normal or low cardiac output.

All the hypoxic patients had pulmonary arterial hypertension which was reduced by inhalation of high concentration of oxygen. All these patients after exercise showed reduction in arterial oxygen saturation with further elevation in pulmonary arterial pressure.

Since Dr. McCann has stressed the importance of carbon dioxide, I thought I would here tell you of some studies done by Whitehead, Draper, Goldensohn and others at the University of Colorado Medical Center on what they have called "Diffusion Respiration." This work adds significantly to our knowledge of what we may call the "pharmacology" of carbon dioxide.

Under standard experimental conditions, if a dog is given enough sodium pentothal intravenously to suspend respiration, sufficient oxygen for its metabolic needs continues to flow inward to the alveoli of the lungs and the animal may live for as long as 90 minutes without the application of any sort of artificial respiration. During the period of apnea and "diffusion respiration," the energy responsible for the inflow into the lungs of oxygen is furnished by what these workers have called the "hemoglobin-oxygen pump (Figure 1)." This "pump" acts in the following manner: as long as blood circulates through the lungs a steady stream of reduced hemoglobin passes through the capillaries in the alveolar walls and picks up and carries away as normally oxygen from the alveolar spaces. The volume of oxygen so removed, however, exceeds the volume of carbon dioxide that coincidentally enters the alveolar spaces from the blood, with the result that the ~~barometer~~ pressure within the alveoli falls slowly but steadily below the atmospheric pressure at the glottis and the ambient oxygen moves under this head of pressure into the airways and down into the lungs.

Due to the absence of expiration, the endogenously formed carbon dioxide accumulates steadily and progressively within the lung alveoli and blood and tissues with ultimately fatal results.

If the experiment is terminated at the end of 45 minutes of apnea, by withdrawal of pentothal and the institution of artificial respiration, the animal will make a perfect recovery (Figure 2). For the success of the experiment and the survival of the animal, certain conditions must be met:

1. The animal must be previously de-nitrogenated, that is by breathing 100% oxygen for 45 minutes his body must be largely cleared of nitrogen gas. The presence of nitrogen in the dead-space air is followed by its accumulation in the alveoli during apnea and this interrupts pressure relations -- smothers the pump with a blanket of inert gas.

2. The airway must be kept open at all times. Obstruction of the free flow of oxygen leads to rapid absorption of this gas from the alveoli and rapid collapse of the air spaces.

3. The circulation through the lungs must be adequate to carry away the needed oxygen.

Important metabolic changes taking place and their influence upon the circulation may be summarized as follows:

1. Absence of respiratory movements and their influence on circulation of the blood
2. Extreme respiratory acidosis
3. Marked hemo-concentration
4. After 30 minutes progressively increasing hypoxia
5. Over-dose of pentothol

The following are the important observations on the circulation:

1. No significant or consistent changes in heart rate.
2. During the first five minutes a decided and consistent fall in systolic blood pressure followed by a gradual return to normal. With re-establishment of ventilation of the lungs, the blood pressure rises rapidly

to a level significantly above normal.

3. Electrocardiogram changes. Frequently during the first 30 minutes of apnea extra-systoles appear. In every case these extra systoles disappeared as apnea proceeded and the pH of the blood fell and the level of pentothal sodium diminished. It is thought that the extra systoles were due to the large dose of pentothal sodium required to induce and maintain anaesthesia.

Other experiments showed that light pentothal anaesthesia with low pH and high CO_2 do not of themselves have a marked tendency to cause serious disturbances of cardiac rhythm.

Another interesting observation was the prolonged absence of electrical activity of the brain after about 20 minutes of apnea. This was quickly and completely reversible and was not associated with permanent harm.

McCann calls attention to the very alarming symptoms which may develop rapidly in a hypoxic patient given a high concentration of oxygen to breathe. Under certain conditions such a patient will show decrease of cyanosis but clinically he becomes much worse. Coma and convulsions may ensue. This apparently paradoxical situation ^{may} ~~seems to~~ be due to the rapid development of a high grade respiratory acidosis. During the period of hypoxia before administration of a high grade oxygen concentration, respiration was being driven by anoxic chemoreceptors, and the respiratory center was being depressed by a high CO_2 tension. The administration of oxygen removed the chemoreceptor stimulant to breathing and left the patient at the mercy of a depressed respiratory center. Breathing became shallow, CO_2 accumulated to a still higher level, the pH fell and serious C.N.S. symptoms developed. Dr. McCann points out the high C.S.F. pressure which develops in these patients. This observation was made also experimentally by Goldensohn, Whitehead in 1951.

This type of "oxygen poisoning" is due more to the fall in pH with high CO₂ than to any specific effect of oxygen itself.

A further interesting observation was an apparent mainly reflex anuria which developed almost immediately after or simultaneously with the apnea.

Further significant observations were:

1. As apnea proceeded and carbon dioxide increased, less pentothol was needed to keep the respiration suspended.

2. A small dose of barbiturate reduces or abolishes the distressing effects of ventilation of human persons and animals breathing high concentrations of carbon dioxide.

I direct your attention particularly to the possibility of the precipitation of atelectasis under clinical conditions. Not only a part of the lung may collapse but even an entire lung may collapse if obstruction of an important portion of the airway occurs in the lung in patients who have been breathing high levels of oxygen. I refer you to ^{Engel's} Holmdahl and Risholm's article^{*} of 1951. These men report a fatality from massive atelectasis of the remaining lung during a lobectomy for pulmonary tuberculosis and ascribe it to the mechanism to which reference is made in these remarks. Dr. Whitehead made ~~some~~ other interesting observations. Dogs were tested with diffusion respiration at various barometric pressures. Duration of life at the lowest barometric pressure, that is at the highest altitude, was shortest. The duration of life increased slowly as barometric pressure was increased, that is as the experiment was tried at lower altitudes. Longest duration of life was at sea level. As barometric pressure was increased above atmospheric pressure at sea level, duration of life did not increase.

Legend

Figure 1. The Hemoglobin-Oxygen Pump. The animal has been de-nitrogenated and now breathes only oxygen. As oxygen is removed from the lung, the alveolar pressure is reduced and oxygen flows down the airway. Meanwhile, CO_2 is accumulating. (Anesthesia and Analgesia, Nov.-Dec., 1949)

Figure 2. Carbon dioxide content and pH of arterial and venous blood. During "diffusion respiration" venous CO_2 content and pH show reversal of their normal relations with the arterial. After resumption of ventilation, normal relations are restored. (Anesthesia and Analgesia, Nov.-Dec., 1949)

Footnote: Figures 1 and 2 are reproduced through the courtesy of Anesthesia and Analgesia.

* H. Enghoff, M. Holmdahl and L. Risholm, Oxygen Uptake in Human Lungs without Spontaneous or Artificial Pulmonary Ventilation., *Acta Chirurgica Scand.*, 103, 1952, fasc. 4.

THE HEMOGLOBIN-OXYGEN PUMP

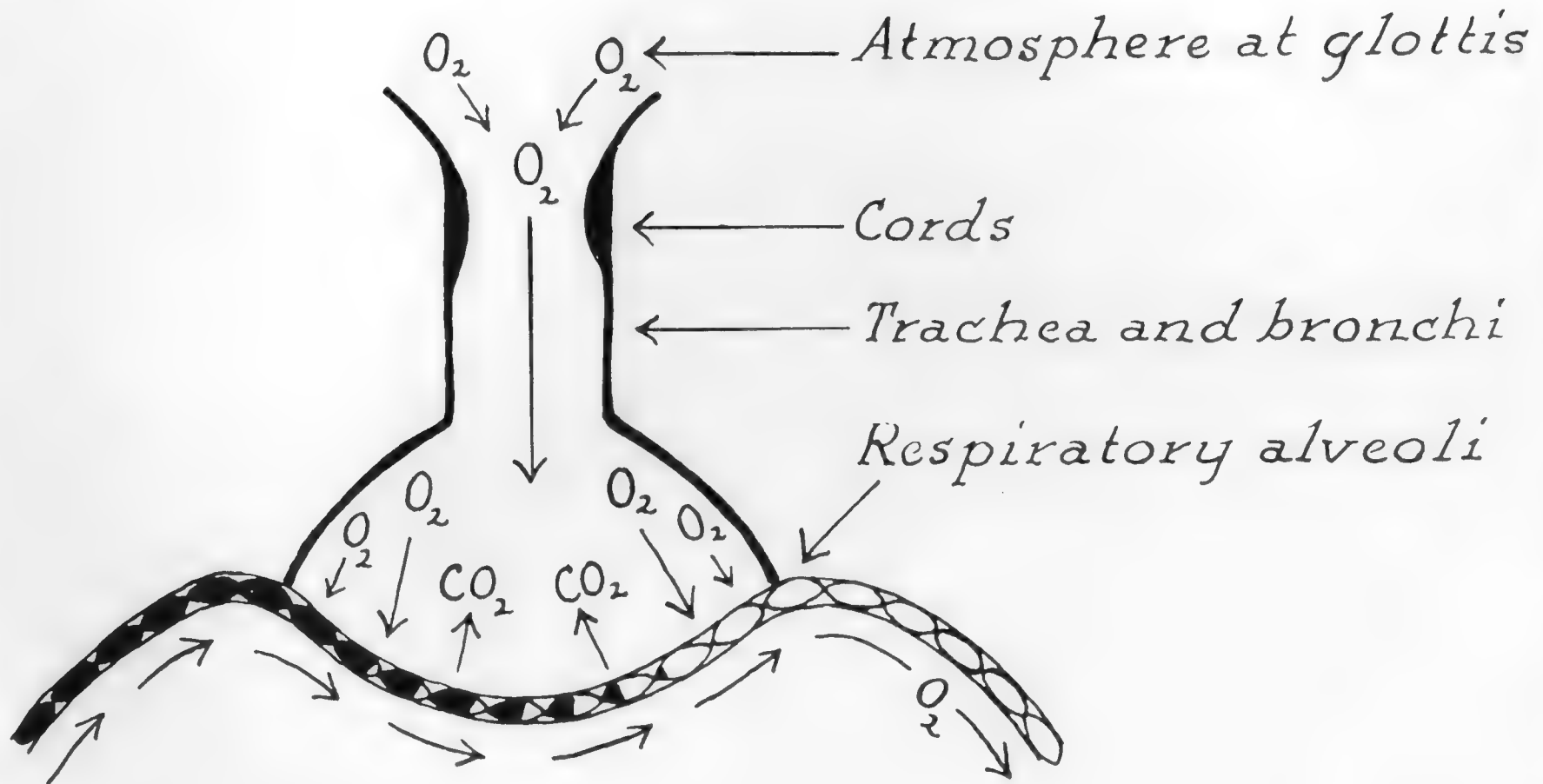


Fig. 1

Diagram of the Caries paper
in the lining



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"Anesthesia and Analgesia - Nov. - Dec. 28: 307-312, 1949.

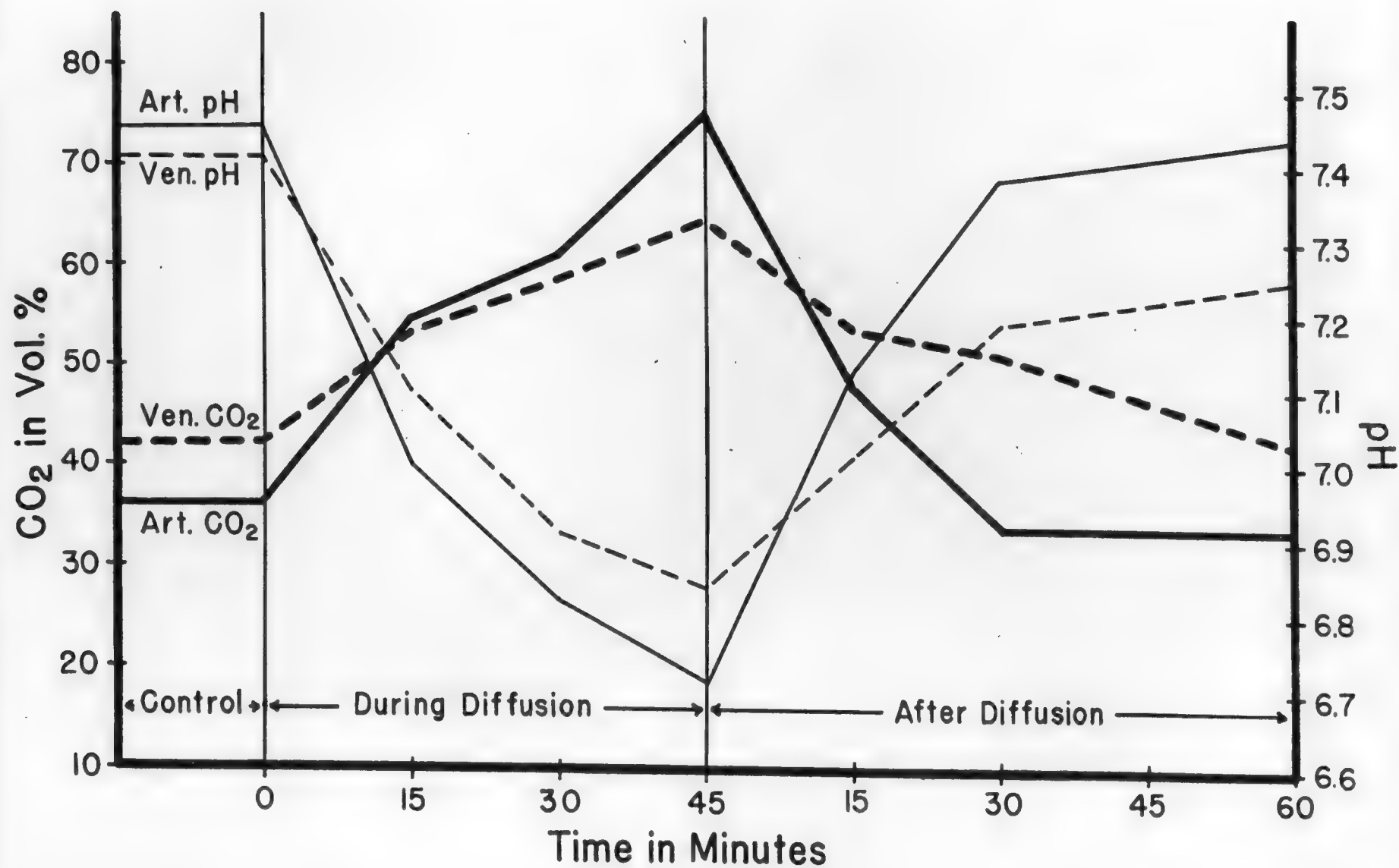


Fig. 2

Microscopic view of T. T. Spring

Photography by
Division of Visual Education
Univ. of Colorado Medical Center
400 E. 17th Ave.
Denver 10, Colorado
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Lister, who was invited to attend a graduating exercise of one of the great universities in the British Isles, and he gave a magnificent address and at the end of his address, there was some demand from the audience that the resident professor of medicine should get up and make a few remarks. Being a modest gentleman, he was reluctant to do it, but the demand increased and finally he stood up and he said, "Ladies and gentlemen, when the English nightingale sings, all other birds should be still". Now when this Rochester nightingale, who is both physician and physiologist, sings, I think the Rocky Mountain magpie, perhaps, ought to shut up.

Now, he has emphasized many important things, and I can only touch on them in just a few moments. He indicts heavily emphysema and the physiological changes that accompany emphysema and I agree with all that. He sings in praise of carbon dioxide and I think very wisely, and also very cautiously so. He emphasizes that in past times, we have emphasized too much heart failure and too little lung failure, and one surely will have to agree with him on that.

Now, at the University of Colorado, we have had competent physiologists and bronchologists and since Doctor McCann was kind enough to send me a copy of his address to try and digest at the higher and more rarified altitude of the Rocky Mountain regions, this denser atmosphere within

and without, why I am grateful to him for letting me have that and I wanted to tell you perhaps something about the formal pharmica dynamics of carbondioxide as it has been studied by Doctor Whitehead and Doctor Draper, Doctor Goldenson and other men in the Departments of Physiology and Pharmacology at the University of Colorado School of Medicine.

So, may I have that first slide, please? Now, in 1908, Volhard talked about what he called 'oxygen pumpers' and the experiments which I will tell you about here will demonstrate something of this.

Now, if you will look at the slide, you will see that we have the ambient atmosphere outside; we have an airway depicted and we have the respiratory alveolae, and moving from left to right, we have the pulmonary circulation in the capillary bed.

Now, if, as was done by my colleagues, if an animal is first de-nitrogenated, that is if he is permitted to breathe one hundred percent oxygen for forty-five minutes until the nitrogen is pretty thoroughly washed out of his system, especially his respiratory airways and if then he is given pentothal of sodium intravenously until his respiration ceases entirely and if he is now continued in a one hundred percent atmosphere or as the circulation proceeds through his capillary bed, oxygen is picked up out

of the alveolar spaces and carried away and carbon dioxide comes into the alveolae. His respiration has ceased entirely under the influence of pentothal of sodium, but because the uptake of oxygen volume exceeds the indigenous CO_2 in the alveolar space, why a valent of pressure is created from the space to the alveolae and the oxygen which is alone in this animal's ambient atmosphere, the atmosphere then flows from his glottis down through his trachea and into his alveolae, is picked up and carried away, and the dog, without any artificial breathing whatsoever, and Doctor Whitehead calls this the fusion restoration rather than oxygen pumper as Vorhard described it, the animal will live without any form of artificial respiration just by virtue of this hemoglobin oxygen pump.

The oxygen is taken away with sufficient rapidity in the alveolae to set up a current, to set up a radium of pressure from the atmosphere down into the lungs, and the animal will continue to survive for forty-five minutes.

Now, he will die after that time if the experiment is continued for the reason that the carbon dioxide elimination gradually blankets the alveolar capillary membrane so that the oxygen is no longer able to move into the - into the arterial and capillary into the blood.

Now, the animal would die if he was immediately taken out from this and put in air, because then there would

be a great inflow of nitrogen and the nitrogen would serve in the same blanketing way and he would die very quickly.

Now, if, after say, say after twenty minutes the carbon dioxide has begun, has been steadily increasing, no longer is pentothal of sodium needed after twenty - between twenty and thirty minutes, because the carbon dioxide accumulation, as Doctor McCann referred to, is sufficient at this time and has an oxidizing influence upon respiration and for the last, say fifteen minutes or longer of the experiment, the animal is not breathing, but he does not require any longer pentothal of sodium to keep him in this state.

Next slide, please. Now, this slide shows you here up above the blood pressure and the picket fence here is the spirometer reading and the movements on the left here show the respirations up and down and then the pentothal sodium is given in sufficient quantity to stop his breathing entirely and that's where you see, over on the left, the point where the up and down respiratory movements quit.

Each one of the large moving down of the lines simply indicates the point at which the spirometer bell is filled again. The moving down simply means the point where the spirometer bell is filled in with oxygen; over here are the respiratory movements, and these figures here do not refer to minutes. These are the minutes down here, but

these refer to certain events in what's happening here.

Now, you can see, although respiration has quit here, oxygen is continued to be taken up by this oxygen hemoglobin from the bell. The bell is filled again, oxygen continues to be taken up, the bell is filled again, oxygen continues to be taken up, and so on.

At this point up in here, you will notice that he had a little fall in blood pressure and then a little rise and then here, this is a run of X-ray cystolic contractions which Doctor Whitehead showed very nicely with - were due not to fall in blood pressure and not to fall in carbon dioxide, but to too much pentothal, and then you notice that they disappear and the blood pressure falls slowly over here and after some sixty minutes over here, why with complete lack of circulation, there is complete stoppage of uptake of oxygen from the bell.

Next slide. Now, he showed very nicely in this slide here, time in minutes in here and oxygen saturation of the hemoglobin is here and here is the arterial saturation and the veinous saturation, the control period. This is during the period of abnema, of - during diffusion and this is after the period of abnema. You notice the period of arterial saturation maintains remarkably well for thirty minutes here as hypoxia develops. You notice the veinous saturation does the same thing and then very rapidly

at the termination of the experiment which is simply terminated by giving artificial respiration with oxygen.

Next slide. Now, this slide shows time in minutes down here, control period here, the apnea appeared here, the after apnea aperiod here, and the CO_2 in control percent here. You will notice that the arterial PH during the accumulation of carbon dioxide, and arterial PH falls, the venous PH falls and the arterial and venous CO_2 both rise quite remarkably. The PH falls down in the neighborhood, in the average around to 6.69.

The next slide, please. Now, here an interesting thing was done. This is similar to the other slide I showed you. There was a little rise in blood pressure here at the beginning of the experiment and a little fall. This is the respiratory movements here. This is where the apnea appears, or that period begins, but the oxygen uptake continues. The spirometer bell was filled again and the oxygen uptake gains again.

Now, at this point right here, the bell was held so that the bell could not fall and monometers were placed so as to record the negative pressure that would be created under these conditions, and you can see that this pressure fell quite remarkably here and amounted to about fourteen centimeters of water. In other words, the oxygen uptake by the alveolar capillary system, under these conditions,

and you can see that this pressure fell quite remarkably here and amounted to about fourteen centimeters of water. In other words, the oxygen uptake by the alveolar capillary system, under these conditions, was sufficient to create a negative pressure in the - or in the airway amounting to umpteen centimeters of water.

Next slide. Now, they subjected some animals to carbon dioxide inhalation, and Doctor McCann referred to this work of Doctor Stott, and there is one in our clinic out there, that work of Doctor Stott, and that was he emphasized the importance that should be attributed or the significance attributed to the fact that the emphysematous patient has a remarkable tolerance to CO_2 . He lives in an atmosphere of low oxygen and a high atmosphere of CO_2 .

Now, if you give this emphysematous patient carbon dioxide to breathe, he establishes it remarkably well until a point where he comes to a critical level. Perhaps Doctor Stott reported that when the CO_2 given to breathe was around about 9.28 percent, the emphysematous patient was remarkably comfortable, but when he was given carbon dioxide to breathe as high as 11.28 percent, he was very extremely uncomfortable.

Now, one other thing in relation to that, and that was this, both in these animals and in the human beings who were subjected to ventilation with high percentages of carbon

dioxide, Doctor Whitehead found that a small dose of pento-thal, while the CO_2 was being breathed at high concentration, could show amazingly the discomfort of this high CO_2 ventilation. A perfectly normal person breathes a high CO_2 concentration, why he becomes very quickly very uncomfortable and this discomfort is very easily controlled by the very small dose of pentothal sodium.

Doctor Whitehead found, in addition, that during the high concentration of CO_2 , there was more barbiturate apparently taken up in the central nervous system than when there was low CO_2 and that suggests the clinical danger perhaps of a patient who has been given, for surgical purposes, a barbiturate and then that patient comes back to the ward and becomes anoxic because of an obstructed airway, and now, very quickly, he has a combination of high CO_2 and esthetic effect, and a high barbiturate can drive or can be driven into his central nervous system to add to his difficulties.

Now, one other thing in relation to this, and that is this, that when a patient breathes oxygen, a higher percentage of oxygen, I should have mentioned this earlier in one of the slides, when this patient breathes a high percentage of oxygen and he gets an obstruction of the airway, why there is no nitrogen, there is only a very small amount of CO_2 there early, and he can get very quickly a complete

collapsible lung or a collapse of a lobe or a segment of his lung, simply because the residual oxygen is only there and that is very quickly absorbed under these conditions.

Now, here you see the effect on a human being of breathing nearly ten percent CO_2 , twenty percent, thirty and forty percent CO_2 and the remarkable effect on the PH breathing these different percentages of CO_2 .

Next slide. Now, experimental slide of treating psychotic patients with CO_2 and with this type of experiment. In other words, it was thought that maybe that inhalation of higher CO_2 and the dropping down in PH might have an important influence upon the underlying abnormalities within the central nervous system that made a patient schizophrenic or at least so abnormal that he couldn't get along with the rest of us, and so they tried this on a number of patients and found that within a small number, that they were apparently helped by breathing this high percentage of CO_2 with a change in their PH.

Now, they found also one other thing that was very interesting and that was that practically immediately the apnea ensues, the kidneys stop secreting urine and when the experiment was terminated and the respiration starts again, the kidneys immediately start secreting urine again, and they determined that this was a reflex by denervating one kidney by injecting a local anesthetic around that kidney, and

leaving the other kidney without any anesthetic around it, and they found there that the normal kidney not denervated by local anesthetic continued or stopped secreting immediately that the abnea was started. The other kidney that was denervated, why, continued to secrete urine, but at a lower rate than normal, and after the experiment was terminated, and a period of abnea was over here, why, the denervated kidney rapidly returned to a higher rate of secretion of urine and the one that was not denervated, why, resumed also its normal rate of secretion.

Now, I have reported this because I thought that these might have mono-dynamic influence or effects of carbon dioxide might bear an important relation to what Doctor McCann has been telling you.

(Applause).

BY DOCTOR VORWALD:

May I beg your patience just for a few moments. It is twelve o'clock and there is only one announcement, if you remember, the photograph which was taken yesterday. There will be a young lady in the foyer of the building for most of you who wish to have one. I am sure she will satisfy your needs, and so with that, let us be off to lunch.

(Session adjourned at 12:00 noon)

Discussion

DR. MCKENNETHER: I am greatly honored to be invited to attend this erudite meeting which has now, by virtue of the diverse and inspired work of my old friends, the late Dr. LeRoy Gardner and Dr. Vorwald, become an international event.

Now I feel foolhardy speaking after the most stimulating papers of this morning, but I will do my best and shall not detain you long.

All I have been able to do in the short time available is to bring up to date our mortality and pathological data on this matter and lay them before you in the confident hope that if there is an explanation of them and there are deductions to be drawn from them, I shall obtain them during this conference.

Frankly, I am puzzled and disturbed at the possibility which may emerge, not only in connection with the question of an etiological relationship between cancer of the lung and asbestos, but also between cancer of the lung and other local irritants.

In 1949, I published a note of the deaths from asbestosis, asbestosis with tuberculosis, and either of these also with cancer of the lung, recorded in the United Kingdom from 1924 to 1947. I placed alongside them for comparison the corresponding data we have from silicosis and cancer of the lung. These were factual data which speak for themselves insofar as they go, but I attempted to make no deductions, since I felt the asbestosis deaths were too few for the purpose. I have now brought these figures up to date.

K2-2

The corresponding figures for silicosis deaths amount to several thousands. They were, I think, sufficiently large to show that there is no etiological relationship between silicosis and cancer of the lung and in this I ^{am} fortified by the data produced by the South African experts. I will not weary you, therefore, with the additions in the last three and a quarter years to our silicosis figures.

Briefly, therefore, our latest figures for recorded deaths from asbestosis, asbestosis with tuberculosis, or either complicated with cancer, are 306. I am excluding ten of these where there is also cancer of sites other than the lungs. Our net figures are, therefore, 296 deaths and of these forty-eight or 16.2 per cent were associated with primary cancer of the lung. This shows an increase from a 13.2 per cent disclosed by our earlier figures. This means that, as more deaths from asbestosis come to hand, the cumulative percentage of deaths with complicating cancer of the lung is rising rather than falling.

Now, where do we go to from here? Are these figures sufficient to indicate a casual relationship between the retention of asbestos dust in the lungs and subsequent cancer of the lung? The small numbers certainly dictate caution. Nevertheless, they represent the great majority of the deaths from asbestosis which have occurred in the United Kingdom during the past quarter of a century.

Nowadays, because of the greatly increased awareness of workers, doctors, and others in the neighborhood of asbestos plants, of the

disease; because of the duty of coroners to investigate with autopsy and to report to my department deaths suspected to be due to occupational disease, whatever it may be; because of the compensation benefits accruing to cases of pneumoconiosis; because of the system of factory inspection in force; because of our periodical check-up of the death certificates provided by the Registrar General and for other reasons, it is likely that very few deaths from asbestosis escape notice.

Therefore, with confidence, I think we can say that the figures point to the necessity for the collection of similar data from other communities with an asbestosis hazard, so that by means of statistical examination of the global figures so obtained we can settle the question of whether cancer of the lung is an additional risk of asbestos exposures.

I would remind you here that these figures include all the deaths from asbestosis, during the period in question, which we can trace and which have been verified pathologically. Undoubtedly, the deaths of some cases, which to my personal knowledge had developed asbestosis round about 1930, have escaped notice, for reasons about which we can only surmise: they may have emigrated or died from other causes, including primary or secondary lung cancers, and thus the asbestosis and occupational factors have passed unrecorded. For the reasons I have mentioned, however, such deaths have been increasingly unlikely to escape the net during the past 20 years.

K2-44

I mention this more particularly because of the real danger of duplication of cases in compilations of global figures from different countries. Often there have been reports in the United Kingdom of cases or of series of cases from different observers of particular aspects of the disease, and often the same cases have appeared in more than one of the reports. There are no duplicates in the figures given here.

There is another point to be borne in mind and that is that the population at risk from asbestos is small relatively to other industries and it fluctuates. A war brings a great increase of people at risk, because obviously there is a great demand for high class asbestos insulation for use in warships and other types of work. Then when peace comes the demand drops off again, so that one can never really isolate a population, for instance, to which one could relate particular cases of asbestosis, assuming that asbestos is a factor in this matter.

We must be careful, therefore, as I have implied, that we do not accidentally magnify the risk. On the other hand, it is possible for a risk to be concealed because of an erroneous estimation of the population exposed to the risk. Thus, as I found a number of years ago in the coke-oven industry, only a small proportion of those employed are exposed to tar or tarry vapours, and in them the risk of cutaneous cancer is about six times that of the general population. Yet if one relates the cases to the whole population of the coke-oven trade, the risk is

entirely submerged. In other words, the risk is concealed in the overall numbers erroneously assumed to be at risk.

And we shall have to consider this point in relation to asbestosis and cancer of the lung; a considerable proportion of workers in the asbestos industry have very little exposure to asbestos - on the other hand, we do not know whether much or little exposure to asbestos is required to trigger-off the cancer of the lung, if, in fact, there is such a relationship.

This morning Dr. Lynch asked, "What is the degree of asbestosis?" In fact, all these people have been autopsied and all of them died primarily of asbestosis, whether they had tubercle or cancer also.

I will not weary you with the statistical approaches we have made to our slender data, but will content myself with a rough breakdown of the figures.

Of the 296 deaths we are considering, 172 occurred in males and 124 in females. Eighty-two or 27.7 per cent, half males and half females, were associated with pulmonary tuberculosis. The mean age at death was 48.2 years, with a range of 24 to 73 years for the cases of asbestosis; 39.3 years with a range of 17 to 66 years, for the cases of asbestosis with tuberculosis; and 53.4 years with the range of 32 to 77 years for the cases of asbestosis and cancer of the lung.

The mean duration of exposure was 15.7 years with a range of 1 to 48 years for deaths from asbestosis; 11.1 years with a range of 1 to 33 years for deaths from asbestosis with tuberculosis,

and 19 years with a range of 2 to 42 years for deaths from asbestosis with cancer of the lung.

The mean duration of life between cessation of exposure and death was 6.5 years with a range of 1 to 32 years for deaths from asbestosis, 4.7 years with ^Arange of 1 to 21 years for deaths from asbestosis with tuberculosis, and 5.8 years with a range of 1 to 26 years for deaths from asbestosis and cancer of the lung. You see the ranges are very wide indeed, which is of some interest. Now, there are indications, from our data, that our preventive regulations which came into full operations in 1932, twenty years ago, are having an effect and this may be the explanation of why the incidence of complicating cancer of the lung appears to be rising.

Today, we do not encounter cases of extreme massive fibrosis and gross bronchiectasis and even anasarca and a 'nutmeg' liver with death (uncomplicated with tuberculosis or cancer of the lung) at an early age, within 5 to 7 years from the commencement of exposure. Those cases do not seem to occur. The duration of exposure which results in death from asbestosis is lengthening, the age of death is increasing and the degree of fibrosis found at autopsy is less.

This means that cases of asbestosis are living to the cancer-occurring age period, and if - and I say if - there is a relationship between asbestosis and cancer of the lung and if irritation - let us surmise from the asbestos fiber-is a co-carcinogen with a long lag

152-7
period, then we would expect an increased incidence of cancer of the lung over that found in the general population to become evident.

Regarding these cancer cases, the mean age of death, as I said, was 53.4 years, and the range 32 to 77 years, and the mean duration of exposure 19 years with a range of 2 to 42 years. Now, the occupations involved may be interesting, but I would certainly not attempt to draw any deductions, since several occupations with different dust exposures were -- and to a lesser extent nowadays are -- carried out in the same room. Nevertheless 10 of the 48 cases were associated predominantly with weaving, 8 with disintegrating, mixing or opening fibre, 7 with pipe and boiler covering, and 6 with mattress making -- all very dusty jobs in the earlier days.

Well, what then is the etiological factor, if any, here? I would be most grateful for your views. I know that physical irritation as a direct carcinogenic agent has been rather discounted by the cancer experts, but I remember that a United States expert, Dr. Oppenheimer, has done some experimental work with cellophane wrapped round the kidney, which may be a pointer.

DR. RHODES: Dr. Lynch, was there no correlation in your series between the extent of asbestosis and the occurrence of neoplastic change?

DR. LYNCH: The three cases of carcinoma in our series occurred in advanced cases of asbestosis, not in minor conditions where a diagnosis is made merely because asbestosis might be present.

DR. LANZA: I find it difficult to believe that the cancer incidence among asbestos workers in the United States could be anywhere near as high as it appears to be in England. We may have missed some cancer cases but I do not believe we could have missed so many if they had occurred to the same extent they apparently do in England. It is true, of course, that as soon as the harmful effects of asbestos were recognized by American industry a program of cleaning up the plants was instituted, and although all asbestos plants in the country have not attained perfection in industrial hygiene, great strides have been made, particularly in the plant in Charlotte, Dr. Lynch's city. With the number of workers in the industry so small, it is obvious that we shall never have the clinical experience with asbestosis that we have had with silicosis.

I used to hear frequently in Canada that the Canadian asbestos is less damaging than the kind used in England where the major part of the asbestos used, I am informed by my English friends, is Rhodanian asbestos, a variety quite dissimilar in structure and physical characteristics to the Canadian kind. I am told also that there are differences in the Canadian varieties, between the asbestos mined at the town of Asbestos and the kind mined not far away at Thetford. Since experimental studies in the past few years have shown that different types of silica may produce entirely dissimilar pathological effects, I am beginning to wonder whether we have given sufficient attention to differences in the type of asbestos in our attempt to explain variations in the rate of asbestosis.

KL-9

DR. SMITH: I had an opportunity to visit Dr. Glyne shortly before his death and to learn something about the material he had assembled in England. He had 17 cases in which there was co-existent asbestosis and carcinoma of the lung. In his series there was no detectable relation between the degree of asbestosis and the presence or absence of cancer. His series came from a plant that used the Rhodesian blue asbestos which, as Dr. Lanza has pointed out, has certain characteristics that are different from those of Canadian asbestos. The Rhodesian variety has a more brittle fiber and, therefore, gives rise to more dust. I understand, however, that some of the British cases had been exposed to Canadian asbestos.

Earlier the question of carcinogenicity/nickel was brought up. Recently, I visited Dr. Amor, formerly medical officer for the plant in Wales where the nasal sinus tumors had been observed, and Dr. Morgan, who is the medical officer there at present. They stated that the men who developed the nasal sinus tumors had not been employed in the part of the plant where there might be exposure to nickel carbonyl. Both doctors were very reluctant, therefore, to believe that nickel carbonyl was a factor in the nasal sinus tumors observed. They pointed out that the tumors which had occurred were in men exposed to arsenical dust from the calciners and that cancer of the nasal sinus was not seen in men who were employed in the industry only after 1924, when the calciners were re-designed.

DR. RHOADS: Dr. Merewether, would you comment on these remarks?

KL 71
DR. MARGARETH: I am afraid I cannot comment on all of them, but I think I can partially answer some and that Dr. Knox can answer others.

First of all, I think Dr. Lanza is misinformed about the proportions and kinds of asbestos used in England. The original cases of asbestosis, fifty years ago, were all from Canadian asbestos. The earliest commercial asbestos work in England was in the early seventies of the last century and there may have been some Italian fiber used here then, but very quickly the trade concentrated on the white Canadian chrysotile - namely a hydrous magnesium silicate.

Later on, the Rhodesian chrysotile, the South African blue crocidolite, and another South African type, amosite, also mainly an iron silicate, came into use. Now, of all of these, amosite, the brown long fibered material, is the most brittle and dusty - to such an extent that its use in England has declined. I do not ^{believe} they use any blue there at all and they have had a number of cases of cancer of the lung, as one might expect, of course not all the cases of cancer of the lung, even if there is an association with asbestos, can be ascribed to it because there must have been an ordinary incidence of cancer of the lung irrespective of this possible factor.

If Dr. Knox were here, he would be able to tell us about his plant, which is the largest in the country, about the types of asbestos they use, and about his experience of cancer of the lung. The fact is that mainly chrysotile is used in his factory.

KL-61

Concerning the so-called nickel cancer, it is quite correct that none of us think that nickel carbonyl has anything to do with it as such.

Dr. Amor, Dr. Morgan, and myself feel that the re-building of the old calciners had a crucial effect on the risk, although there was some change in the constitution of the semi-refined matter shipped from Canada. The difference -- as I know, because I saw them before and afterwards -- between the old and extremely dust calciners and the modern practically dustless building is enormous.

DR. LEVIN: Dr. Knox informed me that in the plant under discussion there were from 1932 to 1951, exactly 16 cases of lung cancer. Unfortunately, Dr. Knox did not have sufficient data to enable him to say whether or not that was an excessive number of cases.

WORKMEN'S COMPENSATION -- MEDICAL AND LEGAL ASPECTS

Chairman; M. W. Thompson

2:30 to 5:30 P.M.

Concepts of Disability Under Workmen's Compensation Statutes

Theodore C. Waters

Compensation for Disability and Death Resulting From the Pneumoconioses

George W. Wright, M. D.
George E. Meredith
Martin F. Hilfinger

VORWALD COLL
BOX 89-91

Discussion, led by

Andrew Kalmykow

&

BY DOCTOR VORWALD:

Ladies and Gentlemen, may we begin. We have a very tight program for this afternoon and we will have to adhere as closely as we can to schedule.

Now, if you refer to your programs, as you undoubtedly have during the first three and a half days of this Symposium, you will note, I am sure that there are two broad areas which are covered. The first area has reference to medical phases and the second area has reference primarily to the medical-legal aspects in compensation.

During the first three and a half days, guests, you have heard Jenny Lind from Boston, you have heard the oriole from Baltimore leading off from Doctor Waring, and

you have heard the English sparrows, and I discussed this with Doctor Orenstein. He said there are no singing birds in South Africa, but we are very fond of the ostrich, so - and, yes, we've heard the nightingale from Rochester and the magpie from Colorado and no matter how beautiful their song might have been or how comforting or discomfoting, I know that many of you who have sat through this three and a half days were medical men, and you've had some distress parts, various parts, and the men from the legal profession, compensation field, sometimes, I'm sure in sympathy, other times with a rather high degree of forbearance, but now it is their turn and you medical men, physiologists, psychologists and psychiatrists and what not, must sit and listen to them. So I think they are now in a position to do to you what you have done unto them.

Now, much of this program, particularly the last day and a half, would have been impossible had it not been for the great help and counsel of the co-chairman of this Symposium, Mr. Waters. Mr. Waters has been a long friend of the Saranac Laboratory and the Trudeau Foundation and I want, for the record, to say again that the success which this symposium may have is, in large measure, due to his effort.

Mr. Waters comes from Baltimore and he is to act as General Chairman for the next day and a half, and he will

also introduce the speakers, and so with that, Mr. Waters.

BY MR. WATERS:

Thank you, Arthur. I know that I speak for the participants in the program this afternoon and tomorrow, when I thank Doctor Vorwald and the Saranac Laboratory for the opportunity to participate in the deliberations of this Symposium. We're all tremendously interested. We've appreciated the information that you doc's have given us. We would like to perplex you with some of the problems that are incident to the administration of compensation laws dealing with the pneumoconiosis. We feel that we have learned a lot. We know that we will learn more before the sessions close. And we hope that you, in turn, will find out something about the point of view of the lawyer who is trying to do the job in handling claims dealing with this type of industry.

Now, I'm going to be very brief in these introductory remarks, because this afternoon, it is my pleasure to introduce to you the chairman who will preside at this particular session. I can only say that he is a grand person and an industrialist. He is a little guy, but a big man and comes from an industry that has concerned itself and has done something about the protection of the health and the welfare of the men that are exposed in that particular industry. Mr. Thompson, from Hall China Company, Mr. Mike Thompson.

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PART SIX

WORKMEN'S COMPENSATION - MEDICAL AND LEGAL ASPECTS

Chapter Twenty-six

Introduction

M. W. Thompson

The object of this symposium, as stated in the program, is to improve industrial health. Following that lead I would like to say just a few words about industrial medical programs in small plants. I believe this topic has a direct bearing on workmen's compensation, the subject to be discussed at this session of the symposium.

First of all a small plant, or a group of small plants, in a so-called hazardous industry cannot really improve its Workmen's Compensation position without an intelligent medical program. Then too, it has been my experience that if a State Industrial Commission knows that a certain small plant or group of small plants in any industry is honestly trying to eliminate hazards and handle intelligently its industrial health problems the members of the Commission will give to that plant or industry a more favorable consideration than they would if the management were not making a proper effort to solve its own problems. The members of the Commission are human and if they know that management is doing its best, their attitude is going to be a friendly one.

Small plants in the United States comprise over 3.6 million establishments, employing almost 19 million workers. When we realize that only a small percentage of these plants or of their employees is experiencing the constructive benefits of on-the-spot in-plant health services, the urgency of the problem becomes apparent. We don't need to bother very much about

the industrial medical programs among the larger manufacturers. Generally they are well taken care of, but the small plants - and I come from a small-plant industry - do need more and better information and more intelligent leadership along this line. Fortunately, many organizations are doing just this. I just want to mention a few organized efforts along this line.

The National Association of Manufacturers made a very comprehensive study in 1951 of industrial health, medical and safety practices. This study will be available in printed form in a few months. As a matter of fact, the study has been completed. It is a very voluminous volume but it will be condensed and will be available for general distribution within a very few months. Comparison of the previous study made in 1940 with this new information shows us that a tremendous advance has taken place in those eleven years and the comparison definitely indicates that industrial management is becoming more and more aware of its problems and responsibilities. That is one agency.

In the industry with which I am connected we have received invaluable help and guidance from the Industrial Hygiene Foundation of Pittsburgh, not only in studying and evaluating the hazards in our industry but also in setting up a proper medical program. The Council of Industrial Health of the A.M.A. has developed and will soon have in print a pamphlet or handbook, entitled Guiding Principles for Small Plant Industrial Health Services, which will be available to all and which will be particularly valuable to management and to general practitioners who devote some of their time to industrial medicine. I understand that the National Industrial Conference Board in New York at the present time is making a study of industrial health programs which will be published in the near future.

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These developments indicate that today, more than ever before, employers in small plants realize that better industrial health means lower workmen's compensation costs, increased production, better employer-employee relations, proper placement of employees and the practice of preventive medicine, all of which really produce greater profits. This enlightened viewpoint, together with the assistance from the agencies mentioned and from many other organizations working along the same line, can result only in more and better industrial health programs.

BY MR. THOMPSON:

Thank you, Ted, for those kind words. You know I'm going to talk, in the few words that I say this afternoon, about something that is not perplexing at all, not along the medical lines or along the legal lines, so I hope you'll bear with me. I am neither a doctor or an attorney. I just try to be a plain businessman.

The subject for our discussion this afternoon is Workmen's Compensation -- Medical and Legal Aspects, and we're very fortunate in having as speakers on the program gentlemen who are tops in their fields. The team is in good shape and we can look forward to an interesting and informative afternoon.

I notice in the forword on the inside cover of the program that the object of this Symposium is to improve industrial health. That's why we're here, so following that lead, I would like to say just a few words about industrial medical programs in small plants. I believe this has a direct bearing on the subject we are discussing this afternoon, Workmen's Compensation.

First of all, a small plant or a group of plants in a so-called hazardous industry can not really improve their Workmen's Compensation position without an intelligent medical program. Then again, it has been my experience that if a State Industrial Commission knows that a certain small

plant or group of plants in any industry is honestly trying to eliminate hazards and intelligently handle its industrial health problems, the members of the Commission will give more favorable consideration to that plant or industry than if management is not making a proper effort to solve its own problems. The members of this Commission are just human and if they know that the people in the industry are really doing the best they can, their attitude is going to be a little different.

Small plants in the United States comprise over 3.6 million establishments, employing almost 19 million workers. When we realize that only a small percentage of these plants or their employees are experiencing the constructive benefits of on-the-spot in-plant health services, then the urgency of the problem becomes apparent.

We don't need to bother very much about the industrial medical programs among the larger manufacturers. Pretty generally, they are well taken care of, but the small plants, and I come from a small plant industry, do need more and better information and more intelligent leadership along this line. Fortunately, many organizations are doing just this. I just want to mention a few organized efforts along this line.

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In the industry that I am connected with, we found that the industrial hygiene foundation in Pittsburgh gave us invaluable help and guidance, not only in studying and evaluating the hazards in our industry, but in setting up a proper medical program.

The Council of Industrial Health of the A. M. A. has developed and will soon have in print a pamphlet or a handbook. Now, it is entitled Guiding Principals For Small Plant Industrial Health Services, and it is pretty near ready for publication. It's coming along in fine shape. When ready within a few months, it will be available to all interested parties. It will be particularly valuable to management and general practitioners who devote some of their time to industrial medicine.

I understand also that the National Industrial Conference Board in New York is making a study at the present time of industrial health programs which will be published in the reasonably near future. This all indicates that today more than ever before, employers in small plants realize that better industrial health means lower workmen's compensation costs, increased production, better employer-employee relations, proper placement of employees and a practice of preventive medicine, all of which really make for greater profits.

This enlightened viewpoint, together with the assistance from the agencies I mentioned above and from many other organizations working along the same line, can result only in more and better industrial health programs.

Now, from there, we go to the first team, and we've got a fine first team on this afternoon. As you will notice in the program, Ted Waters, who is of the firm of Milliken, Stockbridge and Waters, Baltimore, Maryland, is the lead-off man, and so, Ted, will you take the ball from here.

BY MR. WATERS:

You see, you didn't get rid of me as easily as you had expected. Gentlemen, as Doctor Vorwald has remarked, we have been considering the medical aspects of the pneumoconioses for several days and I know you share with me my own personal feeling of appreciation for the opportunity

to meet in the atmosphere of learning of the Saranac Laboratory and of this Symposium to re-consider the various problems incident to the pneumoconioses.

(Mr. Waters' prepared paper, which is on file with the Saranac Laboratory was read).

BY MR. THOMPSON:

Thank you, Ted. I always thought that Ted Waters knew more about Workmen's Compensation as it effects the various states throughout the United States than anybody else, and I'm quite sure that paper proves it.

Now, the next item on the program this afternoon is Compensation for Disability and Death Resulting from the Pneumoconioses.

Now, on this subject, we have a group of four speakers. Unfortunately, Mr. George E. Meredith is not able to attend this afternoon, but we have two other gentlemen who will more than fill his place, but to lead off in this particular discussion, we're going to have Doctor George Wright, who is Director of the Department of Physiology of the Trudeau Foundation. I know that most of you know that. The only reason I repeated it is because someone might be here this afternoon who was not here this morning, so Doctor Wright, will you please take the stand?

BY DOCTOR WRIGHT:

Take the stand?

to meet in the atmosphere of learning of the Saranac Laboratory and of this Symposium to re-consider the various problems incident to the pneumoconioses.

(Mr. Waters prepared paper, which is on file with the Saranac Laboratory, was read)

PART SIX

Workmen's Compensation - Medical and Legal Aspects

Chapter Twenty^{six}~~-seven~~

Concepts of Disability under Workmen's Compensation Statutes

Theodore C. Waters

During the present week.....

.....

.....to effect cure of these diseases.

CONCEPTS OF DISABILITY
UNDER WORKMEN'S COMPENSATION STATUTES

Read at the Seventh Saranac Symposium
on "Pneumoconioses"
Sponsored by The Saranac Laboratory
Saranac Lake, New York
September 22 - 26, 1952

THEODORE C. WATERS

CONCEPTS OF DISABILITY
UNDER WORKMEN'S COMPENSATION STATUTES

During the present week, we have been privileged to meet in an atmosphere of learning to reconsider the problems presented by "pneumoconioses". Certainly there is no place in this country where that subject has received more conscientious scientific and intensive consideration than here at the Saranac Laboratory.

We have renewed our associations with the memories and teachings of those two grand men of industrial medicine--LeRoy U. Gardner and Arthur J. Vorwald--together with their many associates who have rendered such distinguished service in this field of medicine.

The previous meetings of the symposium have been devoted to the medical aspects of pneumoconioses; beginning with the present session, we turn to the legal aspects of these problems to consider:

1. Legislative provisions for compensation for the victims of these diseases, and
2. The evaluation of disability or injury arising therefrom.

These problems are complex and give rise to differences of opinion, dependent upon the point of view of approach.

In order that I may identify myself for the record, I wish to state that in my professional practice, I have always represented employers and insurers in matters dealing with this subject, except in a number of instances where my employer clients

have requested me to represent their employees in the presentation of claims. While my professional representation has been on the side of employers, my thinking has always been influenced by deep sentiment and regard for those tragic figures who become disabled or die from diseases for which the medical profession has found no cure.

Our brothers of the medical profession have already admitted that they do not know all about pneumoconioses. There are many unsolved questions that should stimulate continued laboratory research of the type and character that has distinguished the activities of this Laboratory. From the legal standpoint, our legislatures, commissions and courts have been wrestling with the legal problems relating to employer liability for compensation; and here again, we find that they have not found all the answers to those problems.

In my remarks, please consider that I am thinking out-loud with no conclusive opinions, but still am one of a group who is trying to find answers to questions that have so perplexed my legal brethren.

Dr. Vorwald has asked me to discuss the legal concept of the term "disability" under the compensation statutes. As a prelude to that discussion, may I remind you of the nature of an employer's liability to pay compensation for these diseases. The liability is two-fold: (1) that imposed by the common law, and (2) that imposed by the Workmen's Compensation Acts of the several states. Common law liability results from the employer's negligence in providing for employees a safe place in which to work, safe tools with which

to work and failure to warn employees of dangers incident to their employment. In such actions the employer retains the common law defenses of:

1. The Fellow Servant Doctrine,
2. Assumption of risk by the employee, and
3. Contributory negligence of the employee.

The cause is tried in a Court of Law, before a jury, and technical legal principles of procedure and rules of legal evidence prevail. The ultimate questions of liability and damages are within the discretion of the jury.

Several decades ago, our society became dissatisfied with that procedure and devised the Workmen's Compensation Acts to provide remedies for that type of industrial injury. Today all of the states and federal jurisdictions have enacted such Acts; they differ in form and in benefits; 4 states⁽¹⁾ do not provide compensation for occupation diseases, and in 21 states⁽²⁾ benefits for the pneumoconioses are subject to limitations that are not placed on benefits for accidental injuries, while in 14 states⁽³⁾ no benefits are paid for partial disability. By the acceptance of the provisions of the Compensation Acts, the employer becomes an insurer of the health of his employees as to those diseases made compensable thereby and loses his common law defenses in such actions, while the employee surrenders his right of common law actions and accepts the Compensation Act as his exclusive remedy of the injury so sustained. In such claims there is no issue of negligence, either on the part of employee or employer and the technical rules of procedure and evidence are abandoned.

Perhaps one of the most troublesome aspects of providing compensation for the dust diseases results from the varying concepts of the term "disability" under the Law. Some compensation laws define that term; some leave it to commission or court determination. Certainly one of the basic purposes of the compensation statutes was to provide monetary compensation for injuries resulting in loss of wage-earning capacity of the injured employee.

The common law concept of the term is that of "injury to the body"; while under the compensation statutes, three distinct concepts of the term have been adopted:

1. Disability implies an inability on the part of the injured employee to earn full wages in the employment in which he was last employed. (New York)

2. Disability shall mean that an employee is totally unable to perform any further work in his then occupation or in any other trade, business or occupation. (Vermont)

3. Disability means the event of an employee becoming actually incapacitated, either partially or totally from performing his work in the last occupation in which he was exposed to the hazards of such disease. (Maryland)

Bearing in mind those concepts of disability, let us consider only briefly the three main special provisions of our Compensation Acts that apply to the pneumoconioses:

1. Limitation Provisions of Monetary Benefits.

2. Limitation Provisions Relating to Filing
Claims after Disablement or Death, and

3. Denial of Compensation for Partial Disability.

Time does not permit detailed discussion of these subjects, but I would like to refer to their purposes and objectives. When laws were first amended to provide compensation for dust diseases, it was recognized that many employees who had been previously exposed to dust had accumulated dust in their lungs over the period of employment antedating the effective date of the law. This created so-called "accrued liability" or "potential liability" because when the law was amended, employers were liable as insurers and deprived of their common law defenses. Therefore, many of the states adopted provisions known as escalator clauses of monetary benefits, providing a low benefit at the effective date of the Act with gradual increases in benefits each successive month thereafter. Such provision was contained in the New York Dust Disease Act of 1936. This served to relieve the immediate impact of liability and to discourage an avalanche of claims as soon as the amendment became effective. This pattern was followed in many states.

While many of the states have retained these legislative provisions, others have since repealed them. The fact is that the employee disabled from dust diseases is just as much entitled to compensation as the employee suffering from other types of disability.

Limitation provisions relating to the filing of claims present a serious question. Employers and their insurance carriers properly desire to ascertain and discharge their liability under the law within a reasonable time after last exposure or the termination

of employment. They wish to avoid continuing liability for an extended time. Our courts have interpreted these statutes liberally, giving to the claimant the benefit of doubt. With that philosophy, I am in accord, but there should be some reasonable time limit within which claims must be filed. However, our courts have adopted the general theory that limitations of time for the filing of claims do not begin to run until the employee knows or ought to know that he has contracted the disease. The result is that claims are filed and allowed years after the employment has terminated. Legally, such doctrines do not make sense. With respect to other types of injuries or in cases of damage actions, all state laws provide some fixed period of time within which claim must be filed or suit brought. Why not as to these diseases? I pose the question, but do not profess to know the answer.

With respect to the matter of compensation for partial disability for the pneumoconioses, a far more serious problem is presented, and the legal profession is awaiting informed opinion from our medical brethren before finding the solution. If, as and when the medical profession can find some satisfactory formula for evaluating such disability, I have no doubt that our laws will be amended in this respect; but we do not seem to have reached that stage yet.

As stated before, the laws of 14 states provide that no compensation shall be paid for partial disability while in 4 states⁽⁴⁾, legislative provisions attempt to provide limited compensation therefor. It is not my purpose to advocate provisions for or against such benefits as we shall be enlightened upon that subject by the

speakers who are to follow, but I believe it will be of interest for me to outline briefly the reasons for this problem and to state the arguments for and against the allowance of compensation for partial disability.

The old story is true that the day we were born, we began to die. With advancing age, most of us show lung changes demonstrable upon x-ray examination. Every employee engaged in a dusty trade may show such markings which the medical profession may attribute in part to his exposure. As a matter of fact, many silicotics are engaged in trade and should be continued in their employment even though they have demonstrable evidence of the disease without impairment of wage loss or embarrassment in the performance of their duties. From the standpoint of the employer, he feels that to allow that employee compensation would mean that he would be on the compensation payroll for life, with the privilege of reopening his claim from time to time with increasing benefits; in other words, every employee is a potential claimant where he has been subjected to any dust exposure. In many instances such employees may continue to perform their regular duties for a normal lifetime without wage loss. But from the standpoint of the employee, he feels that he has suffered some injury--as distinct from the concept of disability; that his disease may later become complicated with some other infection, resulting in wage loss or leading to death; furthermore, if his employment is terminated, he may be denied employment in a similar trade upon physical examination. He may not be "disabled" but he is injured. Recently in a meeting where I participated in a discussion of this subject with Dr. Vorwald

and Dr. Mayer, they used the term "disfunction". Perhaps during the course of their remarks, they may attempt some definition of that term.

In summary, the reasons for denying and granting compensation for partial disability are as follows:

Reasons for Denying Compensation:

1. Impossibility of medical evaluation of the extent of disability.
2. Compensation should not be paid unless the claimant has sustained a wage loss.
3. Employer's liability should be fully determined and discharged within a reasonable time after termination of exposure or employment.

Reasons for Granting Compensation:

1. Employee has received some injury.
2. If employment is terminated, employee may be unable to obtain other employment because of his condition.
3. Compensation for partial disability from other diseases is generally allowed.

Four states have legislative formulae that attempt to answer this problem. They are Maryland, North Carolina, West Virginia and Wisconsin; and I commend their statutes for your consideration. The gist of their provisions is to provide limited benefits for "partial disability", the payment of which discharges the employer from continuing liability. Such methods avoid the imposition of unjust and unreasonable costs upon industry and make

the liability of the employer terminable and insurable; they also give to the employee monetary benefits during the period of his readjustment in other employment where he is removed from continuing hazards.

May I refer briefly to the provisions of the Workmen's Compensation Act of West Virginia, with which I am perhaps more familiar both as to its subject matter and its administration. The law of that state provides as follows:

"Section 6-a. Stages of Silicosis; Benefits and Mode of Payment to Employees and Dependents.

An employee shall, for the purposes hereof, be deemed to have silicosis: (1) In the first stage when it is found by the commissioner that the earliest detectable specific signs of silicosis are present, whether or not capacity for work is or has been impaired by such silicosis; (2) In the second stage when it is found by the commissioner that definite and specific physical signs of silicosis are present, and that capacity for work is or has been impaired by that disease; (3) In the third stage when it is found by the commissioner that the employee has silicosis resulting in total permanent disability, whether or not accompanied by tuberculosis of the lungs."

Compensation payable for first stage silicosis is the sum of \$1,000; for the second stage, \$2,000. Such payment operates as a full release and discharge of the employer for continuing liability. There is no requirement that the employee shall file claim; it is within his election to do so. The purpose of the statute is to provide a fund for his readjustment in other employment where he did not continue to be exposed to the hazard of dust. As a practical matter, this law has worked successfully. Certainly, no state has had more serious problems particularly with reference to dust exposure among coal miners. It may not be the full answer to the question, but

apparently has been administered to the satisfaction of employers and employees.

The foregoing discussion has been presented with the purpose of directing your thoughts toward those questions that are troublesome in the administration of laws dealing with compensation for pneumoconioses. State legislatures will continue to search for proper legislative formulae. Let us hope that our medical brethren may help us to find the answer to these problems and also find some method to effect cure of these diseases.

* * * * *

REFERENCES

Reference (1): Four States

KANSAS, MISSISSIPPI, OKLAHOMA, SOUTH CAROLINA.

Reference (2): Twenty-one States

ALABAMA, ARIZONA, ARKANSAS, COLORADO, FLORIDA,
GEORGIA, IDAHO, IOWA, MARYLAND, MASSACHUSETTS,
MICHIGAN, MINNESOTA, NEW HAMPSHIRE, NEW MEXICO,
OREGON, PENNSYLVANIA, SOUTH DAKOTA, TEXAS, UTAH,
VERMONT, WEST VIRGINIA.

Reference (3): Fourteen States

ARIZONA, COLORADO, FLORIDA, IDAHO, MASSACHUSETTS,
MICHIGAN, MINNESOTA, NEW HAMPSHIRE, NEW YORK,
OHIO, PENNSYLVANIA, SOUTH DAKOTA, UTAH, VERMONT.

Reference (4): Four States

MARYLAND, NORTH CAROLINA, WEST VIRGINIA, WISCONSIN.

Chapter Twenty-eight

Compensation for Disability and Death Resulting from the Pneumoconioses
Remarks by George W. Wright, M.D.

This is a most complex problem. The things.....
.....
.....frequently they cannot do.

DR. WRIGHT'S Paper

BY DOCTOR WRIGHT:

Take the stand?

BY MR. THOMPSON:

Yes, sir.

BY DOCTOR WRIGHT:

I'm not so keen about that.

BY MR. WATERS:

Raise your hand and be sworn, please.

BY DOCTOR WRIGHT:

As a matter of fact, I wasn't too keen about the early remarks of this afternoon. It would appear as though I'm to be drawn and quartered or pilloried in some way. I'm the only medical representative, I believe, and these are supposed to be the lawyers' innings. I'm at least permitted a few words before the final events.

I was reluctant to appear on this program, as I think Mr. Waters would inform you, and before saying anything, I should like to establish my position. There are others in the room who have had a vast experience in the strictly medical aspects of the compensation problem. At first hand, I mean. I've had relatively little. Most of the knowledge that I may possess stems from questions that have been directed toward me and it is by virtue of these that I do have, I believe, some comprehension of the problem faced by the doctor and the problem faced by the legal profession and the problem faced by the administrators.

I would like, most of all, just to be here, to answer questions if I can, so my remarks will be quite brief.

This is a most complex problem. The things that we had to talk about this morning, by comparison, are simple. They are subject to measurement. Doctor Orenstein said to me in the course of some of our conversations this week, one of the great difficulties in the field of compensation is that it turns on a matter of opinion and opinion is difficult to measure. If a legal problem comes up about construction of a bridge, an engineer can go and measure it and can state in legal hearings that such are the measurements of that bridge, and if you don't believe it, get someone else to go out and measure it and it will come out the same way. Usually the information desired from the doctor is not subject to strict measurement.

Many things are involved. Some can be measured, but in the last analysis, the physician still has to give an opinion and an equally able and properly motivated physician may, as sometimes does happen, take the opposite side with a conflicting opinion. It seems to me that so long as we must deal in terms of opinion, we are going to be beset with a man who will take one side and another who will take the other side. I must say Solomon himself would have had difficulty in some of the circumstances that have been presented to me.

Now, I was intrigued by something that Mr. Waters said. I have long looked for a definition that he made in a very easy, offhand manner as though I should have known it all my life. This something was his definition of disability under the common law. I don't know much about law but I do believe the common law is

the result of many, many years of experience. Is that correct?

BY MR. WATERS:

That's right.

BY DOCTOR WRIGHT:

And, therefore, it's very apt to be common sense. Certainly the common law definition of disability is much more common sense to me than is the definition that has been written into most compensation laws.

In compensation law, Mr. Waters said, the definition of disability hinges on the matter of earning power. Who is to measure earning power, and how is one to measure earning power? Many things enter into it that can be determined with some degree of accuracy. There are other factors such as motivation and the rights of a man that cannot be measured but that also enter into an opinion as to whether one has an adequate ability to earn wages. Certainly one of the primary problems that a physician comes up against is this matter of stating whether a man is disabled or not.

To assess disability in terms of "injury to the body" (the common law definition of disability), much more nearly approximates what a physician is trained for and what he has devoted his life to. As physicians, we are used to looking for departures from normal and if disability were defined in terms of injury to the body and not in terms of ability to earn wages, I think that doctors would have a much better chance to satisfy the legal profession and those charged with the duties of administration of these laws.

It makes my day worthwhile to have Mr. Waters tell me that the common law definition is as it is. It appears to me to make sense. It certainly makes more appropriate some of the studies that we have done because our aim, thus far, has been to attempt to disclose and find ways, such as an engineer would use, to recognize departure from normality or "injury to the body." In contrast, when I'm asked to talk about a person's ability to earn wages, I find the task most difficult.

Doctor Orenstein, in his opening remarks at the beginning of this Symposium, made another statement that I thought was very pertinent. I have made this very same statement publicly and in writing. The statement is to the effect that when these compensation laws were drawn up, the lawyers gave the doctors credit for ability that I don't believe we possess. This fact has caused its share of trouble.

BY MR. WATERS:

The docs probably were not consulted, George.

BY DOCTOR WRIGHT:

In the Journal of the American Medical Association in 1949 I published some comments concerning the medical aspects of administering workmen's compensation laws governing industrial pulmonary disease. In going over the problem in my own mind again, my views remain essentially as appear there.

Certainly, a lack of proper understanding of the legal definition of disability has led to much trouble. Many of the men in the medical profession who appear as experts don't know the definition of disability as it is being used in the legal sense. Hence,

the court and the doctor speak of two different things as though they were the same. Confusion is bound to ensue.

When it comes to accuracy of evaluating departure from normal, which is some of the background information on which a doctor must base his opinion, we run into more difficulties. Doctor Fletcher, in his opening remarks, left the impression with me that he would be inclined to define disease in statistical terms; a diseased individual being one who departed more than two standard deviations from normal. Now I should like to show a slide if I may. (Fig. I)

This is a chart showing the distribution of one of the functions of respiration as measured in normal individuals. This is the maximum breathing capacity of a hundred and sixty normal men. I say normal men because we were unable in this group, after searching for it, to find any evidences of what we as physicians recognize as disease. I would like to direct your attention to the enormous spread of this particular ability of the human being. The coefficient of variation is so great that a man might have a fifty per cent loss of this power and still be considered a normal man.

I'll try to make that a bit more clear. Let us say that a perfectly well man was originally a high normal; in other words, up in the top part of two standard deviations. Years later when we examine him we find him down here at the bottom. Such a man may have sustained an injury causing a loss of almost fifty per cent of his abilities but still be in the normal group.

Now, surely, I have either misunderstood Doctor Fletcher or he, I believe, is in error if he is going to attempt to use the statistical approach to define disease in the individual. My main purpose in showing this is to give you some idea of the

difficult position that the doctor finds himself in who is presented with a patient after the alleged injury has occurred, who has no notion of what the man was prior to the injury. The doctor may find him to be in the low normal group. Lacking information as to the man's abilities prior to the alleged injury, the doctor must decide whether or not this man has lost powers down to the low point or whether this low ability is what he was originally endowed by nature with. It is not a simple problem.

May I have the next slide? (Fig. II) I'll show you another example of the same thing. This ^(is) peak work ability, the slide I showed you this morning, and here you see a very appreciable scatter of powers to perform. I showed you another one this morning in which extraordinary athletes were way up here. Normal men possess great difference in their abilities.

It's true that when an individual is so terribly injured that he has very few of his original powers left in any direction; it is then simple to recognize the presence of injury. But when the injury is of lesser degrees, and in some respects this type of scatter defines the degree, the opportunities of clearly recognizing that an injury has occurred are rather remote.

I have put these slides on simply to indicate the extreme difficulty that a physician has in trying to form an opinion that he can be certain about, that a man has been injured, to say nothing of deciding how badly he has been injured.

Now, if it is hard enough to do that on a matter of departure from normal, or what I understand the common law definition of disability is, just think how much more complicated it is to say

whether this man is disabled to the point where he cannot earn wages.

Well, I don't intend to labor that point any further. I'm sure that you're all aware of the tremendous difficulties that we have and I wonder if the time hasn't arrived to perhaps reconsider the ways in which the laws have been written. Perhaps the laws can be brought a little bit closer to the true abilities that the medical profession has to assist you with in these matters.

There is only one other comment I'd like to make and that is the question of causal relationship (between injury and disability) for which the legal profession and the administrators again turn to the physician. Not only must we discover whether an injury has occurred and the degree of injury, but I believe under the law, causal relationship must be shown. Here again the physician is asked for evidence that is not always available at the present time.

The first paper given this morning by Doctor Bristol reveals an attempt to find out whether or not diffuse obstructive emphysema, which can be a crippling disease, is caused by so-called inert or non-specific dusts. This is a very pressing problem of the present day. When faced with a case in compensation involving this situation, who can say with certainty that emphysema is caused by the dust in question. Equally so, who ^(can) say with certainty that it is not caused by it? We simply don't have the critical information.

Almost all of the ailments that I know of which occur as industrial disease can be re-duplicated symptomatically and even to some extent on the radiogram, and (in the case of pulmonary

granuloma of beryllium workers) even at autopsy by naturally occurring non-industrial diseases. It becomes at times very difficult for the medical profession, with certainty, to ascribe a definite demonstrable injury to an industrial origin.

The last plea that I would have to make, Mr. Waters, is for the cooperation of all concerned in a much more intensive study of industrial and related diseases in an effort to get more factual information to disseminate amongst the physicians. They have my sympathy in that they are often so hard-pressed to answer questions that cannot at present be answered. Thank you.

Remarks by Martin F. Hilfinger

Particularly difficult problems.....

.....

.....of other states.

Remarks by E. R. A. Merewether, M.D.

Before 1887 a worker was held to assume.....

.....

.....National Health Insurance Act.

99 100

cf page 567
steno

DR. Merewether's Papers

Remarks by ~~James~~ L. Hill

I was intrigued by Dr. Wright's remark.....

.....

.....might be something else.

Remarks by Andrew Kalmykow

We have just heard.....

.....

.....essential to this end.

Remarks* by Henry D. Sayer

Mr. Waters opened his discourse.....

.....

.....second degree silicosis.

* Paper not read at the symposium on account of illness of Mr. Sayer.

Hilfinger

AS INDUSTRY REGARDS PARTIAL DISABILITY IN DUSTY TRADES

Particularly difficult problems are presented under the Workmen's Compensation Laws by silicosis, asbestosis and other lung conditions resulting from the inhalation, over long periods of time, of certain dusts. These problems are difficult for the administrators of the law from the standpoint of the workers, from the standpoint of the medical profession, the safety engineer, and in a very special way from the standpoint of industry, which in the last analysis must pay the whole cost and try as best it can to collect such cost in the pricing of its products or service.

Notwithstanding that industry has long recognized and pointed out many of these difficulties and has earnestly cooperated in trying to work them out in practical ways, fair both to the workers and to industry, it has been assumed by many to be hostile to any form of compensation for silicosis and unfriendly to the workers in those trades where dust is created. Nothing could be further from the fact. Actually, the record of industry in New York State has been outstanding in working out, with labor and the public authorities, the many difficult obstacles met with in providing compensation for disability from dust in ways and means not too disrupting to the industries involved, thereby avoiding the creation of widespread unemployment.

Characteristically, the original pattern of compensation laws did not include silicosis or other dust disease as a basis for claiming compensation from the employer. For that matter, occupational diseases (of which silicosis is the most frequent and characteristic one) were not directly provided for in the early laws. Probably that fact resulted from the want of a remedy, under the old common law, for disease resulting from the occupation. When compensation laws were

amended specifically to include occupational disease, dust disease of the lungs was not included presumably because of the lack of understanding of the nature and causation of those conditions and the determination medically of them. It took many years of intensive study by medical and engineering researchers to arrive at such understanding. Without express knowledge of the nature of the disease, the kinds and substance of dusts - both as to particle size and concentration -, the length of time of exposure, the effects of frequent changes of employment, the existence of lung pathology without marked effect on working ability or earning capacity, the vast difficulty in many cases of evaluating disability and relating it to the occupation or allocating the disability frequently among different employers by whom the worker has been employed under injurious exposures, lawmakers were moving in complete darkness in attempting to bring such conditions under the law. They were met with the problem of disability. The potential cost of ill-advised laws was frightening, both to industry and to labor. The cost of so-called "accrued liability", by which is understood the cost of full compensation to a worker for a dust disease that became disabling after the law became effective, although years of exposure had occurred, perhaps in the same employment, perhaps in other employments, created a situation unknown and little understood. Employers in trades where dust exposure was a necessary incident of the work could not view with unconcern the burden of possible liability that might wreck their business; labor could not take a chance of closing down the industries on which their livelihood depended and driving them out of the State. And yet, all recognized the necessity of doing something about it to get silicosis and other dust diseases under the law in some form.

Finally, after some years of discussion that got nowhere, the subject was examined by the Industrial Survey Commission, created by the Legislature in 1926, and a bill was drafted to bring silicosis under the law with provision for annual physical examinations of the workers, so that men might be removed from the dusty exposure upon the early manifestations of the disease. Speaking for employers generally, Mark A. Daly, Executive Vice President of Associated Industries, agreed with the Commission to accept the bill; the Industrial Commissioner, Hon. Frances Perkins, agreed that the bill constituted a long step in the right direction. But the representatives of labor notified the Commission that they would oppose most vigorously any bill that made provision for medical examinations as a condition of compensation. The proposal was thereupon dropped by the Industrial Survey Commission. This was long before silicosis or other dust disease was specifically mentioned in any state compensation law. Labor's position, which was understandable, was that the men working in dusty employments and who had no other trade at which they could work, should be allowed to continue their work at their trade, so long as they were physically able to do so. They preferred full wages to compensation. They were insistent, and rightly so, that measures should be enforced for the control of dust. The employers were in complete agreement with that demand, provided the measures of control were practical and were not such as would reduce output to the point where employments in this State could not survive in competition with industries in other non-regulated states.

All of industry, including labor and management, finally agreed, in order to bring silicosis under the law even though in an incomplete form, but with the expectation that benefits would rise as industry got

into the stride, upon a law by which in consideration of no physical or medical examinations, but permitting men to work and earn their regular wages, reduced compensation benefits would be paid upon proof of full disability, that is, inability to work and earn wages at their regular trade; such benefits to increase in accordance with a schedule, as time passed and employment in a dusty trade continued, the amount of compensation being related to the increased exposure under the law; no physical examinations to be required; adequate measures to be taken for the control of dust and the necessary engineering studies to be carried out promptly; and no compensation to be payable for partial disability. That law was enacted in 1936, and since that time benefits have increased, both under the schedule and by agreed legislation, after the top amount set in the schedule had been reached, so that now benefits for full disability equal those for any other condition under the Compensation Law.

It was not by inadvertence that the new law made no provision for benefits to workers claiming for partial disability resulting from silicosis. Such benefits were excluded because of the inherent difficulties in determining and evaluating partial disability in such cases, and because it was deemed essentially unreasonable to charge upon industry, and thereby the buying public, an unmeasurable burden of cost for a condition that is not disabling in the sense that it deprives the worker of his wage earning capacity.

Let us consider some of the characteristics of partial disability, so-called, in silicosis and certain other dust diseases, and why it does not lend itself to accurate determination or evaluation under compensation laws.

First, let me say that the basic principle of all compensation laws is disability or death. By disability is understood inability to work and earn wages. Compensation was provided as a percentage of the lost earning capacity to maintain the injured person and his family. Much confusion of thought occurs when we try to classify as a "disability" merely a physiological fact constituting possibly an impairment of function, but not necessarily constituting a bar to earning full wages. Some are prone to consider a physiological defect the same as a compensation disablement. The New York Workmen's Compensation Law in Section 37, relating to occupational disease, defines a compensable disability as a "disablement" which prevents the worker from earning full wages at his trade or occupation. The condition of silicosis is one characteristic of working for years in employments where silica dust is generated or present. The dust, to be harmful to the human body, must contain particles under a certain microscopic size and in a sufficiently large number. We commonly find workers in such dust exposures showing definite evidence of silicosis, both by X-Ray and by clinical findings, and yet they are working regular hours and earning full wages, the same as those who do not exhibit the same conditions. The doctor correctly testifies that the man has silicosis, but in view of the fact that the person is working and earning full wages, he cannot say that he is "disabled". If he cannot work and earn wages, he is totally disabled, and in any such case he is unquestionably entitled to full compensation benefits - as a life pension if the condition is classified as permanent, and as temporary total disability when the condition is not so classified.

Then again, there is the well-nigh impossibility of evaluating

the degree of partial disability in dust conditions of the lungs. I am told by medical experts that there is no standard and no way of telling by X-ray the degree, if any, of partial disability. Men showing a marked silicosis by X-ray may not have any clinical signs or symptoms of the condition, while other men showing very little or no positive signs by X-ray, may nonetheless have severe complaints and shortness of breath and allege an inability to earn full wages at their occupation. At the stoneyard, cutting and polishing stone, at the moulder's bench or in the foundry, running a rock drill in the tunnel or in the quarry, if men work they earn full wages; the unions see to that, if the employers do not, and there is no light work or partial ability to work. There are only two classes of men - those who are able to and do work, and those who are disabled and are thereby entitled to full compensation.

It has been urged that employers are disinclined to hire new men who are known to have any degree of silicosis, even though not at the time disabled. It has been said that the fear of the cost to them of paying full compensation benefits, in case the man later becomes totally disabled, bars such a man from getting remunerative work. This did not seem to us well founded. Yet, we approved and agreed to a bill, enacted a few years ago, whereby the eventual total disability due to silicosis or other dust disease was deemed to be in the nature of a second injury and so the liability of the employer in such a case is shared in by the Special Disability Fund, to which all employers contribute regardless of whether or not they have any cases that are payable out of the Fund. Since New York State led the way in a constructive and practical manner to meet the problems presented by silicosis and similar conditions, and since in its main features industry has cooperated fully and has agreed with the program, we take

pride in the accomplishments of the New York Legislature.

As I have pointed out, however, the question of compensation for partial disability from silicosis presents difficulties that to us seem to be insurmountable. When attempting to surmount one difficulty, we find that thereby we may increase the difficulties presented by another. It is in this field that we find the necessity of keeping clearly in mind the distinction between a physiologic or anatomic defect or so-called "disability", and the disability that prevents wage earning. The one present physical discomfort and even a lowered vital capacity, without loss of earnings, while the other presents the actual inability to work, resulting in loss of earnings - in other words, an economic loss such as the compensation laws all seek to indemnify. To compensate for a bodily injury which does not involve a reduction of wage-earning capacity, is to warp the compensation system into something approaching damages recoverable by actions at law.

It may be said by those who urge compensation for partial disability in silicosis that this is no different than the fixing of the schedule payment for loss or loss of use of a member. In every case of a schedule loss of a member, however, it is necessary to make a finding that the condition presented has reached its maximum improvement and that it is a permanent loss. Not so with partial disability from silicosis. Instead, we anticipate that the condition will become worse, and if the worker continues in the same or a similar occupation, we look for eventual total disability, which will be permanent.

In considering this problem, we must keep in mind another distinction. Partial disability, following a traumatic injury, occurs

generally following a period of total disability, and the partial disability most often is reduced by the lapse of time and the effect of healing, until finally it disappears wholly. The trend of partial disability in dust cases is the other way; it never becomes less, it can only change for the worse.

Another thing, at what point does compensable partial disability begin? In the case of traumatic injury or of an acute occupational disease, we have a point before which no recognizable compensable condition can exist, that is, the date of the injury. In the case of silicosis, we have a slowly progressive condition. As generally seen, silicosis or asbestosis is found to exist only after the lapse of a long time, frequently many years. The length of time may be a shorter or longer period by reason of the susceptibility of the individual, the kind of dust, the size of particles and the number of particles found in the atmosphere. This may vary from one employment to another and most often does not all occur in a single employment. Just when a worker may show the characteristic X-ray markings of the disease is difficult to discover. Certainly, they will not be discovered until too late unless physical examinations are made periodically. Will a claim be made at the first signs of dust by X-ray, or by a reduced vital capacity of the lungs, and if so, will an award be made? And if made, how will the percentage loss of use be measured? Will an award close the case or may it subsequently be reopened and a further award made? And if then total disability eventuates, will credit be given against the total disability award for the amount or amounts already paid on the partial condition?

Will the employee be benefited by an award for partial disability? I think not. In the first place, once a man has been officially adjudged

to be a sillicotic, will he be employable? Hardly! An employer whose operations create dust will be quite unlikely to give employment to a man who has already been found, by medical evidence and an official decision of the referee, to have silicosis. It is quite likely that the official board may direct that such person shall not return to a dusty trade on a penalty of losing all subsequent right to compensation for a total disability. And in like circumstance, any other employer will hesitate a long time before hiring such an officially stamped sillicotic. Moreover, few men who have worked at a dusty trade for many years will readily be adaptable for work in a new and different trade. Experience has demonstrated time and time again that men do not readily change occupations when they have learned a trade. This fact has been recognized from early times, as witness the old saw that goes "Shoemaker, stick to thy last."

And what of the effect upon the worker who submits to examination and is then found to be in some degree a sillicotic? Would it surprise anyone if such a person became immediately depressed and utterly discouraged? Would he not be likely to feel that he has been condemned to early total disability and death? Will any possible benefit of the law outweigh the seriously depressing effects of the official adjudication of his disease? Doctors mercifully try to spare their patients the knowledge of a fatal and malignant condition when they find one. How many cases are there of persons who take their lives, because of some real or fancied desperate condition? Would the meager compensation benefits, whatever they might be, make the Compensation Law a kindly one by robbing the worker of peace of mind?

There are no statistics available as to the number of men who may

potentially be claims under any law that provides for partial disability for silicosis. There is no possible way for industry to make an estimate, within reasonably accurate figures, of the cost of compensating for such partial disability, except by the slow and laborious process of having all men in certain industries examined by X-ray, which labor has always fought against.

I know nothing about the making of rates for insurance under the Compensation Law, but I can well imagine that this new field of partial disability would create great difficulties in the way of insurance, and would be costly to the industry to the extent of seriously, and I mean seriously, hampering New York employers in meeting the competition of other states.

with the responsibility of directing the activities of the Associated Industries of New York in legal and legislative matters, dealing primarily with industrial relations. Mr. Hilfinger.

BY MR. HILFINGER:

Mr. Chairman, gentlemen and ladies: George Wright mentioned the birth of opinions, and so forth in this subject, and I sat there just thinking of - they were telling about this professor in medical college. It wasn't your college, Mert, that had this class seemingly, this class for two years, and at the final meeting, he was having with them, at the final meeting he was having with them before graduation, he said, "Gentlemen, I've told you a lot of things in the last two years." He says, "I suppose if I said that fifty percent of them are correct, I suppose that would be a good average". He said, "The trouble is, I don't know which fifty percent it is".

I feel very remiss here if I didn't congratulate Doctor Vorwald and Doctor Wright and the Trudeau organization on the work they are doing to solve one of probably the biggest mysteries among the problems that exist today in this field of industry. I feel very much subdued, as I stand here and as I sat here this morning and listened to this group, and I understand that there is representatives here from, I think it's ten or twelve different countries,

all from the medical profession, and the present chairman mentioned here a little while ago, the part that the medical profession is playing in industrial safety and industrial welfare and I congratulate you gentlemen of the profession because industrial medicine, as you know, is just beginning to grow.

You mentioned the small plants, Mr. Chairman. I don't think it will be long before every plant will have a direct connection with some of your profession, and I don't know if you realize it or not, but as just an ordinary citizen to sit here with a group like this and listen to the discussions and see the things that you're doing in these graphs and so forth, which are getting right down to facts, I say, I'd feel remiss if I didn't compliment you and congratulate you on the grand job you're doing to help relieve human suffering and make life a lot more livable, making living a lot better, and I think it's proper to say that the employer today is more and more approaching the subject from that standpoint.

Cost, yes, of course, that's important, but I think behind it all, is the attempt to relieve human suffering, and I think we have made tremendous progress, and I'm sure that it won't be long before you gentlemen in this field will have the answer to the problem we're discussing today.

(The remainder of Mr. Hilfinger's paper was read. A copy has been forwarded to me, and it is attached hereto).

BY MR. THOMPSON:

Thank you very much, Martin. Now, we have heard from three talks on Workmen's Compensation in the United States. It seems to me that it would be very interesting if we had a talk on Workmen's Compensation in other countries, so I'm going to ask Doctor E. R. A. Merewether, Senior Medical Inspector of Factories, the Ministry of Labor and National Services, London, England, if he would be kind enough to tell us about compensation for the pneumoconioses in England.

BY DOCTOR MEREWETHER:

Ladies and gentlemen, you have seen me before, but it's not my fault this time. I'm a stand-in. With a foot in both professions, I have been extremely interested in these papers, and it does illustrate the tremendous difficulties not only intrinsically on the subject, but in different states and different conditions. Just a few words that I have written this morning, which will give some idea of our evolution, is by no means perfect, and it may be in the course of evolution still, and it certainly is not necessarily a model for any different set of circumstances.

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Before 1887, a worker was held to assume all the

risks of his occupation, although he could bring suit for action, to recover for injury and damage. Proof of negligence was difficult, and the high costs made it very expensive. For the fifty years subsequent, there have been legislations imposing duties on the employer of supplying safe working requirements, on the old principal if you keep a dangerous element, you must keep it under control.

Nevertheless, so-called acts of God were remarkably and unduly prevalent and many were the tragic and unjust results.

In 1887, therefore, the employers liability act reached the statute book. The act dealt with accidents only and provided for a measure of compensation which, however, could be advocated partially or entirely on the score of contributory negligence on the part of the workmen. It was not until the advent of the Workmen's Compensation Act in 1906, among the pieces of legislation at that time that compensation for occupational disease, as well as for accidents arising out of and in the course of employment, arrived on the scene.

The occupational diseases and hazards covered were strictly occupation, that is to say practically speaking, they did not occur in the general population irrespective of occupation. They were specifically set out in a schedule to the end, on one side, the disease uprisings, and

opposite, where possible, were detailed the occupations or exposures which commonly caused the disease or poisoning. The aims of this method of prescription, particular prescription, as opposed to general prescription of diseases and possible poisonings, were two-fold: Firstly, to reduce, speed up and simplify litigation, and secondly, by setting up opposite disease or poisoning, the relevant occupations or exposures to - to afford a presumption in favor of the workman.

Thus, the schedule minus the disability, and put opposite coal miner, mining, if a man developed the stigmas which he thought were due to his occupation, he could make a claim, but if he was not a coal miner, to succeed, he would have to prove that his asthmas were due to his work, his particular work.

He - if he was a coal miner, then the burden of proof that the asthmas was not caused by coal mining, was on the employer. This is still a feature of our law today.

The first disease and poisoning to be scheduled in this way, subject to compensation, were anthrax and lead phosphorus and mercurial poisoning. To show you the need for such legislation, with over a thousand cases of lead poisoning noticed in my department, well over a hundred died. Slowly and after full investigation and proof of occupational causation on the disease, the occupational diseases and

positioning were put in the schedule until now there are forty-four on the list including pneumoconiosis and others.

It was not until 1910 that pneumoconiosis was included in the schedule, in form - in the form of silicosis with or without tuberculosis. Total disability only was provided for, and silicosis was then defined as fibroid crisis due to silica dust.

Those were the simple days, and the 1918 act afforded rough justice to worse cases since even with the elementary X-ray apparatus of those days, the slow start of silicotic nodules in the film hit the expert like a charge of machine gun bullets.

By the end of 1925, therefore, the disease the compensation covered, and the machinery for diagnosis quite inadequate and the great new act, the Workmen's Compensation Act of 1925 codified the previous legislation and extended it to partial as well as total disability, also constituted our experts under the Home Office, with your Ministry of Interior to deal with diagnosis and assessment and whose decision was final.

This Board successively described as the Silciosis Medical Board, the Silicosis-Asbestosis Medical Board, and now it's the Pneumoconiosis Medical Board, functions today and its decision on diagnosis questions is still final.

Up until 1930, the compensable dust disease was

still limited to silicosis, now defined as fibrosis of the lungs due to silica dust with or without tuberculosis and with either partial or total disability. Coinciding now with my entry into the Factory Department, then the Home Office, confusion became worse confounded. With our mixed dust elements, it was obvious that the classical descriptions of silicosis following on pure quartz exposures, was inadequate to serve justice. Coal miners' troubles were much in the public eye. The disease I've seen in the pottery and grinding trades did not fit and, to make the situation worse, one of the unshakable foundations of knowledge at that time, that only dust containing free silica, was really injurious, trembled and tottered, as asbestosis loomed darkly as a cloud the size of a man's hand on the horizon.

Many free surveys were set on foot and completed, therefore, between 1925 and 1930, and by the Workmen's Compensation Silicosis and Asbestosis Act in 1930, a law was made, under that act, the range of dust diseases subject to compensation and occupations to be covered were greatly extended. The diseases were now covered by an extended definition, fibrosis of the lungs due to silica dust, asbestos dust or other dust, to be defined, with or without tuberculosis.

Relative calm now set in for a time, but soon uneasiness became apparent, that the coal miners and trimmers

were not getting a fair deal. The slate dressers as opposed to the slate quarriers and some foundry operators, seemed to escape adequate notice and busy nurses; that was a very awkward disease and missed notice. The cotton workers felt they were left out.

Some investigations, on a considerable scale, were set up successively in the coal mines, the whole gamut of foundry work and so on, the slate dressers and some foundry operators being dealt with on existing evidence.

In 1943, this culminated in Workmen's Compensation Pneumoconiosis Act, which provided comprehensively for coal miners and trimmers and cotton workers, also in legislative form. The definition now became fibrosis of the lungs due to silica dust, asbestos dust or other dust including the condition known as dust reticulation of the lungs.

I may say it is now a pity, we are taking evidence as to whether this is quite the right definition. In the meantime, in the Beveridge Report on comprehensive insurance burst like an earthquake on the scene and resulted in a series of acts, Round about 1946, most of them coming into force in 1948, and our new law today.

The one we're most concerned with is the National Insurance Injury Act in 1946, which changed the whole system of what was formerly Workmen's Compensation. Formerly, workmen's compensation was a liability wholly on the employer,

provisions being made with respect to pneumoconiosis for contributions as more required during the previous five years, but even then sometimes it was impossible to tax liability to an employer, so the man didn't get the money; also the acts were adjudicated by the County Court judges with, with appeal to the high courts. Compensation was payable for partial or total disability, but was based on loss of wages with an upper limit as to the amount payable weekly and the total amount payable on the date of the claim.

Under the new act, the industrial Injuries Act, the present Act, compensation or benefits, as it is now called, is payable for loss of faculty, which is not quite the same as disablement, and is given in the form of pension if the assessment is twenty percent or over, and in the form of a maturity if it's under twenty percent. Diagnosis is made as before, by the Pneumoconiosis Board. The assessment is appealable to the Appeal Board, but the diagnosis of pneumoconiosis is not. The equal differences are assigned by an appropriately constituted appeal tribunal and the different interpretation of principals of the law is by the Commission, who is the Queen's Counsel of Distinction, and not by the Courts.

The administration of the mass of claims by safeguards, where our decision is investigated by a non-medical

official called the insurance officer, is helpful. Rate of percentage for total disability is forty-five shillings a week irrespective of earnings with additions to wife and dependents. If necessary, that saves a man's - that means if a man is a veteran, an additional allowance is given.

The basic rate of pension is exactly the same as the basic rate for disabled war veterans, the - and the committee under His Honor, Judge Hancock, of which I was a member, correlated on an exact basis, the rates for what are known as the schedule disabilities. That is to say that if a man had lost a hand as a result of an occupational accident, he would get exactly the same award, other things being equal, as a soldier who lost his hand as a result of a war.

Furthermore, if the injured workmen from circumstances beyond his control, is unable to maintain himself and family, he may obtain under another act, the Public Assistance Act, further financial assistance to prove - on proof of need. Medical treatment is covered by the National Health Insurance Act, rehabilitation, vocational guidance and training and placing for a new occupation is obtained under the provisions of yet another act, the disabled persons employment Act.

Special factories called the granule factories are set up in South Wales to provide employment to disabled

persons mostly miners there. Here there was formerly little alternative work for pharmaceutical factories. Those factories, they were built by the Government, and they get - the firms are induced to go there, suitable firms, and get a very low rate and other arrangements provided they employ fifty percent of disabled persons, not necessarily silicotics.

In there we do see - we do make greatest efforts to keep out the tubercle as we do in all the dust producing industries. To those totally unable to compete in the - to compete in the labor market, even under relatively sheltered conditions, there are the "rent-buy" factories, which are almost necessarily, at the present time at any rate, run at a loss. Thus, no disabled person need beg for an existence and the inducements to disabled persons to make the most of the disability is continued.

Costs of these arrangements are paid for out of a fund administered by the Government and the money is provided by contributions by the employees or the workers with a contribution by the Treasury. The fund is a very large sum in hand at the moment. It was an idea -- I pay about a pound a month is about my share. The contribution is the same for men, different for women, different for young persons.

Now, an insured person, under this act, is anybody

under a contract of service with certain exceptions, not a person under a contract for service.

Now, I've got two minutes, I think, just two minutes, yes, two; two things were raised that were very interesting, by Mr. Waters. The common law of disability: In our other Workmen's Compensation, people were getting very much larger damages by a common law action. Now, he can proceed to take his workmen's compensation and he can still claim against the employer. Should he succeed, his workmen's compensation amount is abated by the appropriate sum. These common law actions are exceedingly interesting and very troublesome, because juries - as one of you, I think it was Mr. Hilfinger, said - that juries were very peculiar in the way they award the damages and then you find that somebody making ten thousand dollars for loss of two fingers, and somebody else made, may get two hundred pounds, and so, however, that is the case and there is a very curious reason.

When the liability was put on employer, of course, there was a certain sanction to take care of, but believe me, the common law actions were even more greater sanction, because supposing a man gets his hand off in a press. Well, necessarily, the Factory Department prosecutes the employer for not guarding the machine. Well, that is - the factory is under criminal law, not civil law, and a successful

prosecution, all the man has got to do in making his claim is to prove the conviction and he can prove the negligence.

Now, the other point is limitation of claims. In the times of the Workmen's Compensation Act, the worker first may have two years to make a claim for silicosis, then it was increased to five years and could not be increased longer, because the firm might have gone out of existence, but under the new act, there is no limit to the time the man may put in a claim for compensation.

Then, the other thing is that when a man, a disabled person, gets another illness, ordinary illness like everybody must get and is disabled with it, he, of course, gets the appropriate, much smaller amount under the National Health Insurance Act. Thank you. (Applause)

BY MR. THOMPSON:

Thank you very much, Mr. Merewether. We're going to take a seventh inning stretch for exactly five minutes, inasmuch as our time is short and will you please be back in your seats in five minutes.

(Recess from 4:35 to 4:45 P. M.)

BY DOCTOR VORWALD:

We'd like to get on with the program, so will you please take your seats; we'd like to get on with the program. We'd like to start, so that there will be plenty of time for you to enter into the discussion.

BY MR. THOMPSON:

Yes, we'll please come to order again. We have only one more talk and then I'm going to ask just a few to say a very, very few words. Our last speaker is Andrew Kalmykow, Assistant Manager of the Casualty Department, Association of Casualty and Surety Companies of New York City. He has specialized in compensation matters, both in trial practice and in field legislation. Mr. Kalmykow.

BY MR. KALMYKOW:

(Mr. Kalmykow read a prepared paper, which is on file with the Saranac Laboratory).

BY MR. THOMPSON:

Thank you, Andrew, that was very well done; I appreciate it. Now, the meeting is now open for general discussion, and with the type of talks we've had this afternoon, it seems to me that there should be a good many questions. We ask you, please, will you be kind enough to direct your question to the speaker who you'd like to have answer that question.

BY DOCTOR BENNETT:

I'd like to ask Doctor Merewether a question. He brought up the question of medical boards and I'd like to have him elaborate a little more on that.

BY DOCTOR MEREWETHER:

I thought the worst had now come. I sat in the

COMPENSATION FOR DISABILITY AND DEATH
FROM THE PNEUMOCONIOSES - SOME COMMENTS

By

Andrew Kalaykov, Assistant Manager, Casualty Department,
Association of Casualty and Surety Companies,
Presented at Seventh Saranac Symposium, on September 25, 1952,
at The Saranac Laboratory, Saranac Lake, New York

We have just heard some interesting papers on the subject of compensation for disability and death from the pneumoconioses. It seems to me most appropriate that at this meeting the legal aspects of this problem should be discussed as well as the medical. The two are so closely interwoven. Certainly in trying to find the answer to important legal questions, further light on some purely medical problems would be most helpful. At the same time, the physician today plays so vital a part in the administration of workmen's compensation and the other legal phases of industrial health, that legal problems, which he must help answer, are of definite interest to him also. It is only at meetings such as this that an exchange of views can be provided to mutual advantage.

This close relationship has been made particularly evident by the speakers this afternoon. Mr. Waters has discussed the meaning of "disability." He has indicated the legal difficulties which the use of this term has presented. Its proper meaning has proved one of the more troublesome problems which we have had to face with respect to the dust diseases. Both legislatures and the courts have attempted to define it, seldom if ever with complete success. While its importance from a legal viewpoint is apparent, it has very definite medical connotations. Some laws definitely place upon Medical Boards or, as in New York, Committees of Expert Consultants, the burden of determining disability and in some instances the date on which it occurs. Medical witnesses likewise might well be presented with this question. Before being able to determine whether disability exists, it would seem necessary that the physician should know what it is the existence of which he must determine.

I may be wrong but it seems to me that a physician might naturally be inclined to take, what may be termed, a pathological approach to the problem. He may have a tendency to determine the existence of disability solely by reference to physiological or anatomic changes. It is interesting to me, therefore, to note that both Mr. Waters and Mr. Hilfinger have emphasized the economic aspects of this question, the existence of wage loss. They have stated, or at least implied, that mere evidence of lung changes should not be sufficient to entitle a person to compensation. There should, in addition, be a wage loss, that is anatomical changes sufficient to affect the ability to work and earn wages. In making any medical findings concerning the existence or non-existence of disability, it might be well if this phase of the problem were kept in mind.

At this point, however, we come upon the difficult problems that have so ably been discussed by Doctor Wright - the difficulty of trying to translate lung changes into an estimate of the loss in ability to perform work. At the two extremes, one, little or no lung changes, and at the other, a far advanced case, the answer may be clear. It is in the more common in-between stages where the difficulties are mainly presented, both medically and legally.

A physician might well say that he cannot be expected to know whether or not a wage loss exists. He can, however, in expressing any opinion on the question of disability, make it clear that he does not have reference to the economic aspects of the question. If a physician or a Medical Board states that there is disability, the Industrial Commission might well feel bound to follow this finding even though no wage loss exists as may be required by the definition of disability in force in that state.

Emphasis, however, should not be placed too heavily on any one of the factors entering into the concept of "disability." Wage loss may occur for reasons unconnected with health. This was particularly evident during the depression.

It was then, most of us will recall, that the "silicosis problem" was most acute. Wage loss alone, of course, should not entitle one to compensation if there is little, if any, evidence of fibrosis and no interference with the capacity to work. Because of the high level of employment today, attempts have been made, sometimes successfully, to collect compensation even though there was no loss in earnings. At the same time, if too much stress is placed on the requirement of wage loss, attempts may be made again to receive compensation where there is wage loss, but such loss is not due to any pulmonary dust disease. This would be particularly true if there should be a period of economic recession.

That this is not mere idle conjecture is evidenced by an occurrence only some three years ago in Michigan. A small foundry suspended operations. In a very short time about 60 claims were filed and more were expected. This constituted a high percentage of the total personnel. Fortunately, the foundry soon reopened and the situation satisfactorily adjusted itself. However, for a time serious concern was felt. A similar situation could occur elsewhere, possibly even on a larger scale.

Mention has been made of two factors in the determination of disability, (1) incapacity to work due to the disease, and (2) a resulting wage loss. The further question presents itself concerning the nature of the work in relationship to which incapacity must occur. Is it incapacity to do any kind of work? Is it incapacity to perform the work in the last occupation in which exposed to the hazards of the disease, or is it incapacity with respect to the last employment? These alternatives all have been used in various statutory definitions. There are some arguments for and against using any one of these alternatives. If one relates incapacity to any specific occupation, there is the possibility that compensation may have to be paid even though the employee earns higher wages in another occupation.

Cases of this type have occurred. At the same time, requiring that the employee be unable to do work of any kind may, if too strictly interpreted produces harsh results. Possibly the definition found in Illinois¹ may be found to be a preferable alternative. There, reference is made to the disability from earning full wages in the employment in which last exposed to the hazards of the disease or "equal wages in other suitable employment." Here again, however, we come to a matter of interpretation. The courts or the Industrial Commission will have to determine just what work can be considered suitable. A practical rather than strictly legalistic approach is necessary. Some similar language has produced strange results. In one state, for example, it has been held that any work unless similar to that performed at the time of injury was not "work of any reasonable character."²

The meaning of disability, while related, is distinct from the question whether compensation can or should be granted for partial disability from the dust diseases, often and perhaps more appropriately referred to as non-disabling silicosis or asbestosis. Mr. Hilfinger has marshaled the arguments against doing so. Mr. Waters has presented the main arguments both for and against. His mention of the system in West Virginia and several other states of paying a specific sum which relieves the employer of further liability is of considerable interest.

I believe that no one today can validly question the desirability of paying compensation to an individual actually incapacitated from working because of silicosis or asbestosis. What may be feared, however, is the unknown but very large potential liability, if compensation were payable for anatomic changes without apparent interference with the ability to work and without a causally connected wage loss. This possibility is not likely to manifest itself during periods of high

1. Illinois "Workmen's Occupational Diseases Act," sec. 1(e), Illinois Revised Statutes (1951) Chapter 48, §172.36(e).

2. *Samata v. Higgins Industries, Inc., La.* (1944) 18 So. (2d) 202, aff'd 23 So. (2d) 45.

wages and employment but may well do so at a time when industry is least able to stand the cost.

The definition of disability is of importance also in another respect. Usually under workmen's compensation laws there are time limits within which claim must be made. These usually run from the date of disability. If disability is defined otherwise than with respect to actual incapacity to work, an employee may well find his claim barred when he presents it. Such a case actually occurred in Missouri but the court rejected the contention and held that actual incapacity to work was necessary.³ However, in another case in the same state the court reached a different conclusion where it was a matter of computing average wages. The court in that case took the higher wage period even though it did not coincide with the date of actual incapacity to work.⁴ Neither of these cases happens to involve dust diseases but the principle is the same.

There is another important aspect of non-disabling silicosis which should not be forgotten. That is the psychological effect on the employee himself. Once labeled a silicosis, once deemed entitled to compensation for this disease, it is most likely that one will have created an invalid well before an invalid actually exists. In fact, in one specific case an employee continued his regular work for 24 years after he first noticed symptoms of some ailment.⁵ He had completed a full normal working lifetime of 49 years and did not make a claim until he had reached the age of 72. It seems to me most doubtful whether he, his employer or society, would have in any wise benefited if he had received his compensation some 24 years prior to that time probably ending his useful lifetime of work well prior to the age he actually did. While this case arose on a question of time limitations the facts seem to me most interesting.

3. Smith v. Federated Metals Corp., No., (1939) 133 S.W. (2d) 1112.

4. Renfro v. Pittsburgh Plate Glass Co., No. (1939) 130 S.W. (2d) 165.

5. Consolidation Coal Co. v. Dugan, Md. (1951) 83 A. (2d) 863.

We have dwelt at length on the question of disability. This subject is no doubt of great importance with respect to the dust diseases. Our topic this afternoon, however, is broader. It relates to compensation for the pneumoconioses generally. In this connection, I would like to comment on the tendency which has become manifest in some states in recent years, notably New York and New Jersey, to repeal special provisions and safeguards relating to such diseases. Of course some of these provisions were admittedly transitory in nature. One can mention, for example, the graduated scale of benefits or escalator clauses. Others, however, provided more permanent safeguards. These include provisions relating to partial disability, time limitations, exposure, etc. In New York, care was exercised to retain at least some of the major of these. In New Jersey a more drastic change was made. The full effects of this change we are yet to feel. Already some of the more difficult medical questions concerning estimates of disability have come up. It may be well to give careful consideration to all phases of the problem before too drastic ^{legal} surgery is performed. It should be noted that these provisions were inserted into the law when the need for them became manifest. Satisfactory experience under the operation of a law should not be ground for change. Safeguards should not be discarded without carefully weighing the effect of such action during times of low as well as high employment.

If special provisions are to be retained, some thought should be given as to the scope of their application. I believe earlier this week some discussion was had as to the meaning of the word "pneumoconiosis." It might be well to give some thought to the conditions which should because of the nature of the disease come within these special provisions. If long exposure is usually necessary before disability manifests itself, if there is question as to the existence of partial disability, if the disability is due to some type of dust, should not the term dust disease be applicable? There have, for example, been a number of cases of

emphysema and other lung conditions in the Buffalo area allegedly due to exposure to grain dust. Should these provisions be applicable to cases of this type regardless of whether such condition can or cannot, strictly speaking, be labeled pneumoconiosis?

It is a great deal easier to ask questions with respect to the pneumoconioses than to answer them. There is need for wisdom and technical information before adequate solutions to many problems can be found. I cannot help thinking of a recent court decision. Here compensation was denied for failure to show exposure to silicon dioxide dust. There was only exposure to silica, the court said in effect. This error was readily remedied on rehearing.⁶ Where legislation is concerned, remedy is more difficult. It must be based on sound principles founded on a firm factual background. The assistance of the medical profession is essential to this end.

6. *Greene v. Pearch*, Ariz., (1950) 218 Pac. (2d) 875, reversed on rehearing Oct. 9, 1950, 222 Pac. (2d) 805.

back deliberately. The decision on a matter of diagnosis, a question of diagnosis as called for in the regulations, is for the expert board of medical men whose existence has been since 1926 and is final.

BY DOCTOR MANGANO:

My question is this, is this medical board now a fact-finding body or an examining body, in other words, are any controversies permitted?

BY DOCTOR MEREWETHER:

It's a pure diagnostic board, examining board, it does periodic examinations, does post mortems, where, when the coroner and a pathologist - we have inquests, you know, on these cases by the coroner and the lungs are retained. If a member of the board is not there and the decision as to what the man died from is for the board and for nobody else.

BY MR. THOMPSON:

Thank you very much, Doctor. To help the stenographer, when you ask a question, would you be kind enough to announce your name, as well as the party to whom the question is directed.

Any other questions?

BY DOCTOR HERMAN:

Doctor Merewether, while you're still available, we've been greatly interested in the administration of the

Occupational Disease Act in Maryland, in arriving at some idea of partial disability in it, specifically in silicosis, and apparently that's a foregone conclusion and a matter of long experience now in your jurisdiction. Will you comment on that, how you arrive at some ideas of partial disability?

BY DOCTOR MEREWETHER:

You know, some of you fellows are awfully difficult. You know, I slipped out to have a quick one, but I don't feel quite well even now.

Years ago, I was Chairman of a medical board administering pensions, in Ottawa, you remember it, and gradually, there was built up a system of assessment which I've no doubt you've done the same yourselves, remunerations for demonstrable divergences for the normal. I mean if you decide a man was seventy percent disabled, lost all his hearing, fifty percent is probably too high and so on, or lost a leg, and then you consider that as the case then, the man's disability in the general labor market, you can discover what really was the proper assessment for a man with what we call silicotic bronchitis and emphysema, not hypertrophic emphysema, and give him an average over a period of time.

Well, now, when this last act came around, where it agreed to have the assessment divided in ten percent, twenty percent, thirty percent and so forth, the members of

the board have a very fair idea as to what his fair percent of disability is. It is true that, with the silicotic, well, you know yourselves, a silicotic has, for instance, intermittent attacks of slight bronchitis and so forth. If he is bad, or reasonably bad, he may pop off in one of h them, but, overall, they don't get so much intermittent attacks until they get very severely disabled.

Well, the board was fairly easily able, I think, to assess these things, ten percent one way or either way. It's one way or the other, which is within ten percent, which doesn't make a great deal of difference and the man can always claim he's got worse. Yes, I agree with that, you know that if you have ever seen a man die, which many of you have, you have watched him over years bedridden and so forth, still intelligent, still thinking of the industry and so forth, as I've watched some managers, for instance, and high class executives; it is a terrible disease to die from. Asbestosis is worse.

Well, it really doesn't matter whether your ten percent accurate or twenty percent accurate, as long as substantial justice has been done. This craving for - for particularity in the executions of life which we attempt to do, particularly research people, which I've never claimed to be, I find a little difficult to apply to the normal operations of life.

(Applause).

BY DOCTOR HERMAN:

I really wasn't thinking in terms of small increments of the kind that you describe. I was thinking in a broad general way, if a man is - if he's - how are you going to arrive at any other degree of disability other than permanent, permanent total, I mean would it be a question of, oh, let us say, of forty percent or seventy-five percent, which after all, is of considerable moment, and in correlary to that, how far can you use any objective means of arriving at that conclusion, or do you derive your conclusions entirely from man's subjective complaints?

BY DOCTOR MEREWETHER:

You know, Doctor Herman, you'll find just as much difficulty in asking questions as I have in answering them, but you're like most doctors, there is a certain amount of medicine, and a certain amount of inspiration, but it's quite a fact, you see, these things are permanent, a thing like silicosis, but are fluctuating, and it doesn't make a man who is assessed at thirty percent, let us say, at one time, is not going to reach a hundred percent seven years later. They will give him an assessment for several years, but if he suddenly becomes worse, say gets this pneumothorax or something, then he goes onto total disability in a few minutes, well, in a few hours, but his assessment would then be raised in response to a new examination. Does that

answer your question? It's the best I can understand it.

BY DOCTOR HERMAN:

Well, it would be a great deal simpler, of course, if this weren't all set within the frame work of law, which is necessarily relatively rigid. If it were a question of, oh, let us say, your feeling for medicine, your diagnostic notions or anything of that sort, it would be a great deal simpler.

BY DOCTOR MEREWETHER:

I understand you, but the procedure of the board is a little different. When you say a little more complicated than appears on the surface, that would describe it. Not less than two members of the board examine the man. Well, now, it may be that they're a little doubtful about some point, in spite of their experience. Then they consult other members of the board in other parts of the country, or London or somewhere and by that means, rightly or wrongly, they arrive at some measure of disablement or the diagnosis, and assess the man accordingly, but it isn't, you know, after you've got eight of these types of cases a day, you feel as if it, as if it's just about time to go to bed, and it's very exhausting as I'm sure you know, and they don't do a vast number; they're overworked and they're far behind with their cases, but they do try and do justice in that way, not? They have a great attitude within the limits

of the legislation that is laid down in the law, which is the definition I quoted to you. The interpretations of those definitions, medically speaking, is investigated in this board of experts. That is, I don't know whether I've made it clear or not.

BY DOCTOR HERMAN:

Well, it's - I have some idea of how your board operates, but I'm not sure that there is anything comparable to it in the experience of anyone in - of anybody in this country. Mr. Waters may correct me on that.

BY MR. WATERS:

I agree with you.

BY MR. THOMPSON:

Gentlemen, are there any other questions?

(No response).

You know, we are very fortunate to have with us here some members from the State Industrial Commissions and Medical Boards of Review. Now, I don't know them all, but I wonder if - if Mr. Hill, who is Chairman of the Michigan State Commission, if he would care to say a word.

BY MR. HILL:

Well, first of all ...

BY MR. WATERS:

Come up to the platform.

BY MR. HILL:

I haven't much to say, I guess. I will say this, I was immediately intrigued by Doctor Wright's remark that, after all, medical testimony is only opinion and, therefore, is not conclusive, or can not be conclusive, but the Workmen's Compensation administrator has got to make a conclusive finding; I suppose that's his job.

My own experience has been that when we were permitted to or were mandated by our act, to send disputed occupational disease cases, that included dust disease cases as well as lead poison or anything else, to an impartial commission for an examination, and their finding was conclusive, absolutely conclusive, on the question of whether or not the man had the disease alleged, that at least half of our job was done right there.

If it was reported he didn't have the disease, that's all there was to it, and his claim was summarily dismissed, so it's, at least we thought, it was much easier to have it, and more convenient, and we got a better - or more as a result, when used, we made through that vehicle, let's say, medical opinion conclusive.

Now, I think there is one other, one other item that I might mention, and that is that compensation for occupational diseases, including dust disease, of course, has created a great incentive, I believe, for employers and carriers to adopt every known preventive measure and probably

it undoubtedly has created the incentive for research in an effort to find other measures to prevent the various diseases.

In our experience, when we first had an occupational disease law, we had, at one time, sixty lead poisoning cases pending at one time. Last year, we didn't have a single lead poisoning case. I asked one of the large carriers whether the companies had quit using lead, and they told me no, they had just found a way to manage to handle it so that it wasn't dangerous. Trichloroethylene poisoning, for instance, when that was under our original act, which was a schedule act, we had any number of trichloroethylene poisoning. That was a sort of a sixty-four dollar word, and today, you don't even hear of it, and I asked, oh, one or two of the representatives of manufacturers, whether or not they used trichloroethylene as a degreasing agent. They said sure, we don't have any cases. We prevent it; we prevent the disease, and so the mere fact that you're paid compensation benefits, they are paid to the unfortunate persons, nevertheless, the fact that there is the potential liability, has made industry and carriers very eager to prevent it, and I think that tremendous good, without any question, has come from the preventive measures, and I think that's an important - or the safety end of it is an important angle that shouldn't be overlooked in a dis-

cussion of this type.

Now, compensation people think they have problems, but I'm amazed at having been around here a couple - only a couple of days, to see the tremendous problems that the doctors have and the - certainly, my little compliment, if I can add it, I certainly want to congratulate them on the tremendous work they're doing in the occupational disease field.

Now, if you want me to keep on talking, I can say that I disagree with this business on payment of compensation for partial disability. I don't think you can, if you're going to give a man, a man compensation merely because he has silica deposited in his lungs, I - I disagree with that. I think that you've got to tie it up with the wage loss and maybe that's a way - maybe I've missed the boat, but it seems that whether a man is working in the same occupation or in another so-called non-injurious occupation, if he is receiving the same wages in both occupations, whether it's for the same employer or for a different employer, I feel that he shouldn't be entitled to compensation, that compensation should be paid strictly on a wage loss theory, and as far as that, unless he's unable to secure employment because of silicosis. That might be something else. Thank you.

(Applause).

BY MR. THOMPSON:

Thank you very much, Mr. Hill.

BY MR. KNIGHT:

Mr. Chairman, my name is Knight, from Illinois, Chairman of the Illinois Industrial Commission.

BY MR. THOMPSON:

Would you care to say a word, Mr. Knight, please?

BY MR. KNIGHT:

Mr. Chairman, Ladies and Gentlemen: I have heard a good deal about medical boards in the various states. If the problem is severe there, it surely just must be severe in the state where they have no medical board. Illinois is such a state.

In other words, the medical testimony produced in the record is produced entirely by partisan forces, the petitioner puts on his medical, pays for his medical testimony. The respondent places their medical testimony in the record and pays for that testimony. Now, I know, of course, this isn't always the case, but in the four years that I have been in that capacity in Illinois, I know of certain doctors that will willingly give ten percent or dollars worth and I know on the other side doctors that are willing to testify that for a hundred percent for only ten percent, should be given.

That presents quite a problem, doesn't it, to the Commission who has to pass upon it, and when none of us are

doctors. Our Commission is made up of five commissioners who sit in appeal. We have fourteen arbitrators in the field, nine arbitrators in Chicago in the State of Illinois Building, with their court rooms there, five downstate in strategic territory. The arbitrators, as you know, hear the evidence, hear the testimony, see the exhibits and then if they feel an appeal is warranted, they appeal it to us as what is known as the Commission.

Two of those members of the Commission are appointed to represent management, and two to represent the employee group. We don't term the thing labor and capital, but that's the make-up of the Commission, and the fifth member, the Chairman, is the independent member. That happens to be my position.

I have seen this old act, both as to workmen's compensation and as to occupational diseases, grow from its infancy in Illinois. In 1911, when the Workmen's Compensation Act was first passed in Illinois, it came within my province to write the last half of that act. I was just a young lawyer and, of course, I knew more law then than I ever knew since and so, of course, I copied largely the New York Compensation Board Act. I called it the Illinois Compensation Board in conjunction with the other lawyers that were working on it, so the nomenclature wasn't difficult. We just called it Compensation Board, and it wasn't until

1918 that it became known as the Illinois Industrial Commission.

Now, seriously, within - I don't want to hurt the feeling of any of you people from Pennsylvania when I say this, but within some twenty-four miles of my desk on South Street, I am told that better than sixty-five percent of all the steel in the United States is manufactured. Now, perhaps Bethlehem and these other Pennsylvania points will disagree with that, but I know that when we take around the curve over into Gary, and consider the amount of silica that's used, you realize, have some idea of our problem. We're endeavoring to work it out with partisan medical testimony, which I say is just as wrong as for the ordinary layman to attempt to solve the problems of law. He might hit it once in a while, but it's entirely unlikely.

Now, I am - this last 1949 legislature, I re-wrote the act at the request of both management and labor, principally to recodify it, because it took a Philadelphia lawyer to tell where to find the law, the sections were interspersed in anything but properly codified. Do you care to hear anything about this? I don't know whether you do or not.

BY DOCTOR MEREWETHER:

Yes.

BY MR. KNIGHT:

You care to hear this. So, we at least recodified

the act with a few changes and that pertained to the occupational diseases act as well. Now, you must bear in mind that, in Illinois, the Occupational Diseases Act is not mandatory. The company doesn't have to accept that act if they don't choose to do so or an employer. That throws a further complication into the thing, and the farmer isn't under the Workmen's Compensation Act unless he chooses to come under that act.

Now, in the matter of pulmonary disarrangements, what we call the shadow land cases, we feel that any help that we can get on this, honest medical help, is of great value to us, and so in this last legislature, I tried my best to get a provision in there appointing a medical board, and if possible, a medical board made up of representatives from both of the political parties.

Now, I say that because our Senate was strongly Republican this last time in Illinois, and where you get one result in the House, you'd get another one in the Senate, and so the whole thing has been very perplexing in Illinois. We have about 2,260 cases filed a month, with us, which runs into 26,000 or 27,000 cases a year in gross, with - out of about 62,000 injured or diseases people in the same year, so that gives you about the percent that's filed and the percent that's settled.

Now, we have every type of industrial occupational

disease that you can think of. As you know, the Ford people have come in with their new plant with some 34,000 people working in that new plant, and then we have all the other large plants with their tremendous foundries and their exposures to every type of industrial hazard.

Now, in passing upon those, we listen to the evidence on both sides. Of course, it's our hope that we can strike an average between the testimony that seems to come in on both sides, but when I first went in there four years ago, I broke the tie in a case with the Telephone Company, where ten doctors had testified on one side and twelve on the other. I read the testimony of the first doctor on both sides. I could just as well not have read the testimony of the other eleven doctors, because almost verbatim, the testimony was the same.

Now, that's appalling, and I don't believe the Medical Society stands for anything of that sort, but by having partisan men give testimony, you put a premium on that sort of thing. Therefore, I am in favor of a medical board made as non-partisan, or as bi-partisan as you can make it, and hoping from that board to get direction, because after all, this touches into the vitals of the whole nation, into the vitals of the citizenry of the State of Illinois and every other state, at least I think it does, and so each year we hope when these cases come in, that we

have decided to drop them, but still we keep trying.

(Applause).

BY MR. THOMPSON:

Thank you very much, and we appreciate it very much. Now, I'm sorry, our time is running short, and as you all understand, the banquet is tonight, and we can't overrun the time. However, there are a few other gentlemen that I'd like very much just to introduce and ask them if they care to, to speak for not more than two minutes.

Mr. Brodkin of the State of New Jersey, with the Commission there.

BY MR. BRODKIN:

Mr. Chairman, the hour is getting very late and what I'd like to say would really take more than two minutes, so if you will excuse me.

BY MR. THOMPSON:

All right, thank you very much for being here. I know that there are two representatives of the Board of Referees of the State of Ohio. Would one of them care to say anything?

BY THE OHIO BOARD MEMBERS:

I have no pearl of wisdom to add to those which have already been offered; thank you.

BY MR. THOMPSON:

Thank you very much. Now, Mr. Herman of Maryland,

I believe is here, just want to introduce you.

BY DOCTOR HERMAN:

Just for the record, Doctor Herman. I do represent a Medical Board and I'd rather not be identified with the lawyers, with all due respect, but ...

BY MR. WATERS:

That's a dirty slap.

BY DOCTOR HERMAN:

But I was extremely interested in hearing the experience of Illinois, because we may present in - presently, in Maryland, be faced with a somewhat similar situation. I don't want to wash any of that linen here, but that's an extremely interesting thing and I think important matter.

BY MR. THOMPSON:

I do not have a list of the members of the Commission in the various states or Referee or anything like that. Mr. Wisely of Utah, I understand, is here.

BY MR. WISELY:

Glad to be here, thanks.

BY MR. THOMPSON:

If there are any others, why we welcome you, and if you have anything to say we'd be very happy to hear it; if not, the meeting is adjourned.

The attached paper of Mr. Henry Sayer was not read at the afternoon meeting due to limitation of time. However,

the attached paper of Mr. Henry Sayer was not read at the afternoon meeting due to limitation of time. However, I think it should be incorporated in the minutes of this meeting.

(Session adjourned at 6:00 P. M.)

COMPENSATION INSURANCE RATING BOARD

125 PARK AVENUE AT FORTY-SECOND STREET

NEW YORK 17, N. Y.

HENRY D. SAYER
GENERAL MANAGER

September 24, 1952

Dr. Arthur J. Vorwald, Director
The Trudeau Foundation
Saranac Lake, N. Y.

Dear Dr. Vorwald:

On account of Mr. Sayer's sudden illness and the fact that the doctor has ordered him to remain in bed for a couple of days, I am forwarding to you herewith two copies of his discussion paper, which is incomplete because it does not take in any of Mr. Hilfinger's remarks.

Sincerely yours,



Florence Lossack
Secretary to Mr. Sayer

Enc.

Discussion by Henry D. Sayer at the
Seventh Saranac Symposium held at
The Saranac Laboratory, Saranac Lake,
New York, on September 25, 1952

Mr. Waters opened his discourse by identifying himself as to his activities. So, I may also say that, while I am of the insurance industry, I am not speaking here as an insurance man or as voicing the views of any particular group of the industry. On the contrary, may I say that I have been identified for nearly forty years, in numerous capacities, in the administration or working out of problems in workmen's compensation. I was appointed by the Governor as a member of the Industrial Commission and entered upon the administration of that law in 1915, before the law had been a year on the statute books. Ever since that time, I have maintained a contact of some kind and an extreme interest in the system of workmen's compensation. What I say here today, therefore, is borne out of an intimate association with the compensation law and an extreme interest in it. Thus I speak for no particular group.

Mr. Waters referred to the two-fold liability of the employer to pay compensation for dust diseases: one, the liability imposed by the common law, and two, that imposed by workmen's compensation laws. That is entirely proper, for we should understand the nature of the differences in the two systems. I think in this discussion of the legal aspects of the problem, we should keep definitely in mind the liability imposed by workmen's compensation laws, first because some of those laws are compulsory, as for instance New York's, and the others are elective, but with such penalty for non-election that they may be regarded as practically compulsory. He has rightly pointed to the common law defences to any action under employer's liability, among which is the assumption of risk. Silicosis and asbestosis are characteristic

risks of employment in dust of either substance, and with that risk it has been assumed, in the absence of express statutory provision to the contrary, that the employee under the common law in effect waived any right of recovery of damages due to such cause. That brings us right back to workmen's compensation laws, much of the argument for which was the hardship and injustice due to the archaic common law defences.

Damages were a characteristic of actions at law under the common law; compensation for loss of earnings or earning capacity is the outstanding fact of statutory workmen's compensation laws. When, therefore, we speak of partial disability resulting from dust, where no loss of earnings is yet involved, we are really speaking of damages as distinguished from compensation for disability. There are many implications involved in such thinking. Can we have a system based in largest part upon disability compensation and at the same time partly based upon damages, without the danger of breaking down the system we have so carefully erected over the years? If we yield to the arguments of those who would compensate for non-disabling silicosis, where will we stop in the matter of compensating for conditions not related to earnings or earning capacity? Be assured that once that point is yielded, you will experience great difficulty in preserving the compensation system.

The compensation system was arrived at clearly by the acceptance by employers of a liability limited by the terms of the law itself and based without regard to fault as a cause, while at the same time the employers in effect yielded to a liability as certain as daylight. But while assuming such a certain and limited liability, the employer yielded up his former right of having his negligence proved before there could be any recovery, and permitted the abrogation of his ancient common law defences of fellow-

servant's negligence, assumption of the inherent risks of the employment, and the defence of contributory negligence. While accepting the liability of the compensation law, he assumed, as he had a right, that the burden of cost of the compensation system would not be so great as to drive him out of business.

Mr. Waters very clearly set forth his conclusions relative to disability, since this present session is devoted to the consideration of that aspect. He cited the different concepts of disability, as set forth in the statutes of different states. It is important, I think, to keep in mind just what we mean by disability. I should like to point out that the early basis for all compensation was disability, that is, inability to work and earn wages. To be compensable, only those disabilities that were due to accident arising out of and in the course of employment were compensable in nearly all the states where compensation was the law. It had to have relationship to earnings. Consider for a moment, if you will, that all compensation was for a period of time. That period had relation to the inability to work and earn. Then, too, the measure of compensation was the wages of the injured man at the time of the accident or, as in New York, for one year prior to the accident. Why was wages the standard measure for compensation benefits? Solely because that was what he lost. The compensation rate measures the relation to the injured person; the length of time of the payment of benefits measures the seriousness of the injury, representing usually the period of time the man was unable to work.

Mr. Waters has told us of the law and its administration in the State of West Virginia. He tells you that the law works well and is commended by both the employees and the employers. I cannot believe that such a law as he refers to would meet the increasing demand in New York for compensation for

partial disability from silicosis. I doubt that it is any solution to the problem. He tells us that for first-stage silicosis, the State Fund pays \$1,000. Why? First stage is where the earliest detectable signs of the disease are found, but with no effect on the working capacity. Is \$1,000 needed for that man's relief? I doubt it. Is it any compensation for the man to be officially declared to be a silicotic? Is the certain knowledge that is brought home to this man worth \$1,000? Will it "tide him over the hump" till he gets established in some other occupation? If he is a coal miner in West Virginia, it is not going to be easy for him to get any other kind of remunerative work that will maintain him and his family - at least not in that mountainous and coal-producing State. Does he perhaps take his \$1,000 and travel to another State and get a job as a coal miner?

For the second stage, where capacity for work is or has been impaired, the Fund will pay \$2,000. I should expect that there would be few claims indeed for the first stage, the worker preferring to wait for the double rate, or try to make his case appear to be a \$2,000 case. Here again what, if any, economic advantage is it to the silicotic worker? I can see none; but I can see where it might be an advantage to the coal producer to have his liability to that man fully discharged by his acceptance of the meagre compensation for either first or second degree silicosis.

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LEXINGTON 5950

ADDISON E. MULLIKIN
ENOS S. STOCKBRIDGE
THEODORE C. WATERS
JAMES J. McGRATH, JR.
JOHN S. HEBB, III
THEODORE C. WATERS, JR.

October 1, 1952

Dr. Arthur J. Vorwald, Director
The Trudeau Foundation and
The Saranac Laboratory
P. O. Box 551
Saranac Lake, New York

Re: Saranac Symposium - Legal Sessions
Thursday Afternoon and Friday Morning

Dear Art:

I believe that all the participants in the program left copies of their papers as presented with the stenotypist. I gave to her a copy of my remarks on the subject of "Concepts of Disability Under Workmen's Compensation Statutes" and have in my possession a copy of the papers of Mr. Hilfinger and Mr. Genholts. I do not have a copy of the papers presented by Miss Donlon, Dr. McGee, Mr. Barnako, Dr. Braun and Mr. Tiernan. If for any reason you need copies of any of these papers, I would suggest that you write directly to the participants in the program or I will do so at your request.

This will dispatch my responsibility in connection with the Thursday and Friday sessions; but if I can be of any further assistance to you, please let me know.

Sincerely yours,

Theodore C. Waters

Theodore C. Waters

TCW: BH

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H2B*

*Do not have paper
H2B*

all the papers were substituted for

LAW OFFICES

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THEODORE C. WATERS, JR.

October 1, 1952

Dr. Arthur J. Vorwald, Director
The Trudeau Foundation and
The Saranac Laboratory
P. O. Box 551
Saranac Lake, New York

Re: Saranac Symposium
Panel on Workmen's Compensation

Dear Art:

At the time of the conduct of the Panel on Workmen's Compensation, I believe that each participant handed to the stenotypist a copy of his remarks. I have a copy in my own files; and if the stenotypist does not have them, I would be glad to send these copies on for her use.

For her additional assistance, I am enclosing herewith copy of my introductory remarks at the Symposium.

You will recall that Mr. Andrews was not able to be present and Commissioner Otto Wisely of Utah substituted.

no paper in file

With respect to the question and answer period, I would like very much to have the opportunity of editing that discussion so that no errors may be made with respect to the remarks of the participants.

Sincerely yours,

T. C. Waters

Theodore C. Waters

TCW:BH

Enclosure

stenotypist's notes

Discussion

DR. BENNETT: Dr. Merewether, will you elaborate on the subject of medical boards?

DR. MEREWETHER: The decision on a matter of diagnosis, as called for in the regulations, is for the expert medical board that has been in existence since 1926. The board's decision is final.

MR.

DR. MANGANO: Is this medical board a fact-finding body or an examining body; in other words are controversies permitted?

DR. MEREWETHER: It is exclusively a diagnostic board, an examining board. It makes periodic examinations and does post-mortem studies. We have inquests by the coroner on the cases under review and the lungs are retained. The decision as to the cause of death is made by the board and by nobody else.

DR. HERMAN: In the administration of the Occupational Disease Act in Maryland, particularly in cases of silicosis, we have been greatly interested in obtaining some idea of the ~~disability~~ degree of partial disability. Apparently in your jurisdiction, Dr. Merewether, it is a matter of long experience. Will you comment on how you arrive at an estimate of partial disability?

DR. MEREWETHER: Years ago, in Ottawa, when I was chairman of a medical board administering pensions, there was built up a system of remunerations for demonstrable divergences from the normal. If you decide that a man who has lost a leg or his hearing has a disability of a certain per cent in the general labor market, then you can estimate the proper assessment of disability for a man with what we call silicotic bronchitis and emphysema - not hypertrophic emphysema - and can give him an award based upon the average disability over a period of time. When the last act went into effect, in which the assessment is divided into categories of 10 per cent, 20 per cent, and so on, the board members had a good idea of what a fair per cent of a man's disability is.

It is true that a silicotic might have, for example, intermittent attacks of slight bronchitis and, if he is in a poor condition, he might die during an attack, but in general silicotic individuals do not get many

intermittent attacks until they are severely disabled. The board is able to assess these conditions fairly easily, within about 10 per cent one way or the other, and of course the man can always claim that he's getting worse. But it really doesn't matter whether you are 10 per cent accurate or 20 per cent accurate as long as substantial justice is done. Silicosis is a terrible disease from which to die and asbestosis is worse.

OK.
DR. HERMAN: I wasn't thinking of the small increments of disability you mentioned. I had in mind ^a broader, more general view: how do you arrive at a disability of, say, 40 per cent or 75 per cent instead of a total permanent disability, a difference which is of considerable importance? How far do you use objective means of arriving at a given conclusion, or is it derived entirely from the individual's subjective complaints?

DR. MEREWETHER: It is difficult to answer your questions. The estimates of disability are not permanent and they may be changed. A man may be assessed as 30 per cent disabled at one time but that rating does not bar a finding of 100 per cent disability some years later. He may have a certain assessment for several years but if he suddenly gets worse - say he has a pneumothorax - and becomes totally disabled within a few hours, his assessment would be raised in response to a new examination.

DR. HERMAN: It would be much simpler, of course, if all the procedures were not set within the framework of law, which is, necessarily, relatively rigid.

DR. MEREWETHER: The procedure of the board is a little more complicated than appears on the surface. Not less than two members of the board examine the man. If they are doubtful about some point, in spite of their

experience, they consult other members and by that means arrive, rightly or wrongly, at some estimate of disablement or diagnosis and assess the man accordingly. They have great latitude within the limits laid down by law, limits fixed by the definition I quoted. The interpretation of the definition, medically speaking, is investigated by this board of experts.

OK
Herman
DR. HERMAN: I have some idea of how your board operates, but I am not sure that there is any comparable arrangement in this country.

MR. WATERS: I do not know of any similar plan here.

OK
Corked
Knight
MR. KNIGHT: We have heard a great deal about medical boards in various states. If there are serious problems in those states, then surely the problems must be just as severe in states, such as Illinois, that have no medical board. In those states the medical testimony in the record is produced entirely by partisan forces; the petitioner puts on a medical expert and pays for the testimony, and the respondent places his own medical testimony in the record and pays for it. During the four years I have been chairman of the Illinois Industrial Commission I have known of certain doctors who are willing to give 10 per cent. ~~for 100 per cent injury and~~ *others 100 per cent for 10 per cent injury.*(stenotype notes not clear).

That situation presents quite a problem to the Commission, which must pass upon it, since no member of the Commission is a doctor. The Commission is made up of five commissioners, who sit in appeal, and it has 14 arbitrators in the field. Nine of these arbitrators are in Chicago, with court rooms there, and 5 are downstate in strategic territory. The arbitrators hear the evidence, listen to the testimony, view the exhibits, and then, if

they feel an appeal is warranted, refer it to the Commission. Two members of the Commission are appointed to represent management and two to represent the employee group - we don't term the arrangement capital and labor. The fifth member, the chairman, is the independent member. I happen to occupy that position.

I have seen the old act, both as to workmen's compensation and as to occupational diseases, grow from its infancy in Illinois. In 1911, when the Workmen's Compensation Act was first passed in Illinois, it came within my province to write the last half of that act. I was just a young lawyer and, of course, knew more law then than I have known since then. I copied largely the New York Compensation Board Act; other lawyers joined me in working on the act. We just called it the Compensation Board, and it wasn't until 1918 that it became known as the Illinois Industrial Commission.

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I don't wish to offend any of you people from Pennsylvania but I am told that within 24 miles of my desk in Chicago more than 65 per cent of all the steel produced in the United States is manufactured. When one considers the amount of silica used in those operations, some idea of the silicosis problem will be obtained. We are endeavoring to work it out with partisan medical testimony, a procedure just as wrong as an attempt by the ordinary layman to solve problems of law. He might be right once in a while, but it is quite unlikely. At the request of both management and labor I re-wrote the act for the 1949 legislature, principally to recodify it because the sections were not properly arranged. In Illinois the Occupational Diseases Act is not mandatory and neither the employer nor the employee has to accept the act, a condition which complicates matters. Also, the farmer doesn't come under the Workmen's Compensation Act unless he chooses to come under that act.

105

Corrected
by
H. Knight
changes by
H. Knight

In the matter of pulmonary disarrangements - what we call shadow-land cases - we feel that any honest medical help we can get is of great value to us. In the last legislature I tried to have a provision made for appointing a medical board, one made up, if possible, of representatives of both political parties. We have about 2,260 cases filed a month, or approximately 26,000 to 27,000 per year, ^{with a total land} out of about 62,000 persons who are injured or develop an occupational disease during the same year. These figures give you the per cent ^{as in the 2 cases filed and the per cent settled} (stenotype notes not clear). We have every type of industrial occupational disease you can think of. There are some 34,000 persons working in the new Ford plant and we have the many other large plants with their tremendous foundries and their exposures to every type of industrial hazard.

In passing upon these cases we listen to the evidence on both sides and hope to strike an average between the testimony of both sides. In one of my first cases, one involving the telephone company, 10 doctors testified for one side and 12 for the other. I read the testimony of the first doctor on each side. I could just as well have omitted reading the testimony of the other doctors because it was the same, almost verbatim, as that of the first doctor on their side. I don't believe that the Medical Society stands for anything like that but by having partisan men give testimony you put a premium on that sort of thing. I am in favor, therefore, of making a medical board as non-partisan, or as bi-partisan, as possible and I hope to get from that board direction. This matter is very important because it extends into the vitals of the whole nation and of the citizenry of Illinois and of every other state.

DR. HERMAN: I was extremely interested in hearing about the experience in Illinois because we may be faced with a somewhat similar situation in Maryland.

notes by Jane L Hill

Page 101

I was immediately intrigued by Dr. Wright's remark that after all medical testimony is only opinion evidence and therefore is not conclusive and the workmen's compensation administrator must make the conclusive finding. That is our job and generally a difficult one. It would be much easier and we would get a better result if so-called impartial medical findings could be conclusive. In Michigan we were required by statute to send the claimant in all disputed occupational disease cases to an impartial medical commission for examination. Their finding was absolutely conclusive on the question of whether or not he had the disease alleged. At least half of our job was done right there. If it was reported that he did not have the disease his case was summarily dismissed. If it was reported that he had the disease a hearing was had to determine whether he had a compensable disability. It was a convenient method for achieving an excellent result and through that vehicle medical opinion was made conclusive. However, it was for that reason our Supreme Court later held that part of our statute to be unconstitutional.

I think it is quite obvious that compensation for occupational diseases including the dust diseases has provided the incentive for employers and insurance carriers to adopt every known measure and to engage in scientific research to find other measures to eliminate occupational disease hazards and prevent occupational diseases. We had over 60 lead poisoning cases pending at one time after the occupational disease part was added to our Act. We did not have a single lead poisoning case last year. I asked a representative of one of the large carriers whether the employers had quit using lead and he told me, no, but they had found ways and means to use lead so it was not harmful. It was not unusual to have several trichlorethylene poisoning cases. Today we do not even hear of the disease. I have asked representatives of manufacturers whether they

Hill (cont'd)

still used trichlorethylene as a degreasing agent and am told that it is used because it is an excellent degreasing agent. I am also told that there are no cases of trichlorethylene poisoning because safe methods have been devised for using it. That those substances can now be used safely illustrates the value of an adequate prevention program and the result that can be achieved with the proper incentive. I think that tremendous good has resulted from the potential liability under compensation acts for occupational disease. That factor should not be overlooked in a discussion of this type.

I disagree with the idea of paying compensation for partial disability in dust disease cases. I do not believe a man should be paid compensation merely because he has silica or some other dust deposited in his lungs. I don't think a man with abnormal chest findings as a result of such deposit who is working in the same occupation or in another so-called non-injurious occupation without any loss in wages should be entitled to compensation benefits. I think compensation in dust disease cases should be paid on the basis of wage loss caused by either an inability to work in the employment in which he was exposed or because of unemployability due to abnormal lung findings.

The compensation people think they have problems in the dust disease field but after being around here only a couple of days I am amazed at the tremendous problems that the doctors have in this field. I certainly want to add my little compliment, if I may, and congratulate them on the outstanding work they are doing in the occupational disease field.

PART SEVEN

Viewpoints on Compensation for Pulmonary and Other Occupational Diseases

✓
Corrected
Version

Chapter Twenty-nine

Viewpoints on Compensation for Pulmonary and Other Occupational Diseases

Hon. Mary Donlen

My viewpoint as to.....

.....

.....may be humanly possible.

Chapter Thirty

The Functions and Value of Medical Boards and Medical Examiners

in Controverted Compensation Cases

Remarks by Lemuel C. McGee, M.D.

The foreword on the program,.....

.....

.....in workmen's compensation.

Remarks by Edgar Mayer, M.D.

In the state of New York, prior to 1935, certain.....

.....

.....as boards, as time goes on.

Remarks by A. J. Gentholtz

At the outset....

.....

.....of Workmen's Compensation Laws.

VORWALD COLL
BOX 89-91

August 28, 1953

Hon. Mary Donlon,
Chairman, Workmen's Compensation Board,
State of New York,
State Office Building,
Albany 1, N.Y.

Dear Mary,

Thank you for your corrected paper and discussion for the Symposium. The job of editing and the various details connected with publication are going along slowly but surely. At the moment the problems of expense concern me greatly as the publication cost is much more than I anticipated. Either I will have to raise more money or be satisfied with a mere printing job without reproductions of x-rays, microphotographs and various charts.

With every best wish,
yours sincerely,

Arthur J. Vorwald, M.D.

August 28, 1953

Hon. Mary Donlon,
Chairman, Workmen's Compensation Board
State of New York,
State Office Building,
Albany 1, New York

Dear Mary,

It surprises me to know that George Wright has not submitted a report on the project referred to in your letter of August 14th. As I know nothing about this project I would appreciate having from you some of the details especially as they pertain to the funds involved, also the date and nature of this project.

Yours sincerely,

Arthur J. Vorwald, M.D.



MARY DONLON
CHAIRMAN

WORKMEN'S COMPENSATION BOARD
STATE OF NEW YORK
STATE OFFICE BUILDING
ALBANY 1, N. Y.

August 14, 1953

Dr. Arthur J. Vorwald
163 Park Avenue, Box 509
Saranac Lake, N. Y.

Dear Arthur:

On my return from a few days vacation I find your letter of July 27 and I am very sorry indeed to learn that you are resigning as Director of the Trudeau Foundation and the Saranac Laboratory. I shall be glad to know where your work is going to take you after you have completed your commitments in Saranac Lake. I hope you are going to remain in some field where your training and experience will be helpful in industrial problems.

Let me apologize for the delay in returning to you the material sent me with your letter of June 17. I have made a few changes in the tentative digest of the discussion so far as my remarks are concerned, and send it to you herewith. I take it that the stenotype record is not to be published, but only the edited copy.

As you know, I have not received from the Laboratory a report on the project which was directed by George Wright. I think they should somehow get that report into my hands. There must be material in the files that could be written up. Can you arrange this?

With regards and best wishes, I am

Sincerely yours,

Mary Donlon
Mary Donlon
Chairman

MD/if
Enc. (1)

PART SEVEN

Viewpoints on Compensation for Pulmonary and Other Occupational Diseases

Chapter Twenty-nine

Viewpoints on Compensation for Pulmonary and Other Occupational Diseases

Hon. Mary Donlon

My viewpoint as to.....
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Office of the Chairman
Workmen's Compensation Board
State Office Building
Albany, New York
Albany 3-5511, Ext. 333

For Release

Address of Hon. Mary Donlon, Chairman of the
New York State Workmen's Compensation Board,
at the Seventh Saranac Symposium at Saranac
Lake, New York, Friday, September 26, 1952.

VIEWPOINTS ON COMPENSATION FOR PULMONARY AND
OTHER OCCUPATIONAL DISEASES

My viewpoint, as to workmen's compensation for pulmonary and other occupational diseases, is of course the viewpoint of the administrator. As such, I am grateful for the opportunity of participating in this discussion today. In the past, the contribution of workmen's compensation administrators to discussions of the occupational diseases -- outside of their own organizations -- has been much too small. In part, this is the fault of the administrators because they have usually been too busy handling the details of cases to have time also to record and organize their experience. The viewpoints of lawyers and doctors, as to compensating occupational diseases, have therefore often received more consideration -- especially during legislative committee hearings -- than have the viewpoints of administrators.

I shall present my viewpoint in the light of the historical development of occupational disease coverage in the States.

At the start of our major developments in this field, many looked upon occupational disease coverage, and especially the full or general coverage of such diseases, as a leap in the dark. The atmosphere in which discussion took place was that of fear born of ignorance. Speakers brought forth a portfolio of precedents from the experience of foreign countries, and presented this material with an air of overwhelming erudition. Stunned audiences were convinced that the most complicated and restrictive schemes, so impressively described to them, embodied the last word of the world's existing wisdom upon the subject of occupational disease compensation. So it happened that schedules, often double-column schedules, were imported from abroad and sold to the state legislatures. It seemed irrelevant to note that the diseases listed in the schedules often were not prevalent in the particular State which, however, might be afflicted with hazards not mentioned at

all. It was like buying a hat from Paris, just because it was a hat from Paris and regardless of whether it suited the wearer's face.

It happened, however, that in one or two States where the original injury coverage provisions had been left broad and simple, omitting the restrictive word "accident", the courts held that the word "injury" included occupational diseases. Such States therefore started, almost by "accident", with the same compensation coverage of occupational diseases as of accidental injuries. Other States observed the result and liked it. The prevailing pattern in this country now is full or general coverage of occupational diseases. In this respect America leads the world. With almost every session of the legislatures, the column showing the list of States with schedule or restricted coverage of occupational disease shrinks, while simultaneously the column showing the list of full coverage States lengthens.

In every stream there are main currents and counter-currents. Alarm as to compensation coverage for silicosis flared up during the depression that followed 1929, and this alarm started a tidal wave of restrictive bill-drafting, in part applicable to the dust diseases. In consequence many of the State compensation laws are now cluttered with restrictive and discriminatory provisions applicable either to occupational diseases/in general, or peculiar to the dust diseases or pneumoconioses.

Silicosis compensation sometimes is more a phenomenon of unemployment than of physical disability. Large scale unemployment of foundrymen and miners brought on the silicosis compensation scare during the 1930's. Men out of jobs turned to workmen's compensation as the last recourse from starvation. Some of these men received permanent total disability awards. After the war boom in the early 1940's had created an almost insatiable demand for workers and cancelled the customary hiring tabus, many men who had received permanent total disability awards returned to jobs at wages higher than they had earned before. You cannot think straight, as to workmen's compensation for partial disability from silicosis, unless you remember that hiring practices make this issue a hot spot in a good many places.

It has been noted that, as to occupational disease coverage, some of the States started by copying a foreign pattern of schedule coverage. Other States developed an indigenous or American pattern of full coverage, which is now the prevailing pattern in the States. The trend toward simple and comprehensive coverage was complicated by economic counter-currents flowing from the silicosis alarm during the 1930's. Some confusion has, of course, been created by those counter-currents. The orderly and normal progress of occupational disease coverage toward understandable and simple law and administration was definitely interrupted. To clarify our thinking, further comment on the historical background seems indicated.

The history of occupational disease coverage in the States, if and when that history is written, might be entitled "The Conquest of Fear." The first episodes have to do with fear of the monetary cost to industry, so that early preoccupation was with the need for protecting industry from being ruined by supposedly unbearable costs. The legislatures were told that loose practice on the part of administrators in compensating alleged occupational diseases would turn workmen's compensation into "health insurance." Rare or exceptional instances in which questionable awards had been made were seized upon, publicized and magnified out of proportion to their real significance. Calm and well-informed discussion of the problem was sometimes impossible because the opinions of many had been formed by vivid impressions of exceptional cases, rather than by statistical compilations from considerable aggregates of experience. The cost estimates, not anchored to aggregates of mature experience, were sometimes fantastic. The production of a statistical average is often the best, if not the only, cure for distorted thinking both as to performance and cost.

In 1935 the private insurance carriers would not provide coverage, in New York State, for silicosis. Insurance could be obtained only from the State Insurance Fund. The Fund set its insurance rates high and held its breath, waiting for disaster. It was soon discovered that the insurance risk had been exaggerated. Mr. Muller, a former executive director of the Fund, said: "We were afraid of a wolf that was not there." Of course in the absence of a dependable statistical basis for forecasts, insurance

actuaries do feel compelled to make full-sized cost estimates. That is their business responsibility.

When the actuaries had done their work, the lawyers were then called upon to draft legal restrictions and limitations upon compensability. The resulting patterns of occupational disease legislation in some of the States can best be described as barbed wire entanglements. Fantastic restrictions were put in the way of compensability, especially as to silicosis; and sometimes the benefits for occupational diseases were put on a lower level than for accidental injuries, either as to cash payments or medical care, or both. The laws in some States are still cluttered with discriminatory details that are hard to get rid of. Such petty and harassing restrictions are a liability to insurance carriers and employers because they arouse hostility in the ranks of labor, the consequences of which are out of all proportion to the pinch-ponny savings effected by the restrictive provisions. This is beginning to be apparent in the conflict as to compensability of partial disability from silicosis. Such things keep workmen's compensation laws in politics and they should be above political considerations.

In New York, and also in some of the States where the law and administration are well advanced, the second stage of thinking about occupational disease, and especially silicosis coverage, has now been reached. This second stage of thinking on the subject of occupational disease coverage, is marked by preoccupation with an effort to deal justly and without discrimination with all the victims of occupational diseases. The question arises, can we do this, and if so, how? It becomes necessary to try to see the problem in human terms.

When occupational disease problems are seen in human terms, appropriate action may be expected. Some of the difficulties of coverage may be obscure because the flood-light of research has not yet been turned upon them. On the other hand, some of the remaining tasks for legislatures can be approached through demonstration. In a certain agricultural State, there had been persistent and formidable opposition to occupational disease coverage. Committees had reported adversely on the proposal.

There had been the usual negative arguments. It was insisted in the first place that there was no occupational disease problem in that State, and in the second place that the cost of occupational disease coverage would be ruinous. Such a juxtaposition of conflicting and incompatible arguments is a not uncommon phenomenon in hearings before legislative committees. When at last the proposal for coverage reached the floor of the legislature, the proponents of coverage asked permission to furnish a demonstration. Permission was granted. A parade then started. A group of women employees who had contracted dermatitis while working in fruit and berry processing establishments marched down the aisle, holding out their tortured hands for observation. Full coverage of occupational disease was immediately and unanimously adopted by the legislature.

One of the most perplexing of the unsolved problems in many States is that of compensating partial disability from silicosis. This is the case here in New York. There is a dearth of information based upon comprehensive administrative research. Two hypotheses are encountered. One is that there is no such thing as partial disability from silicosis. Another is that the administrative problems of handling partial disability from silicosis are insuperable. In the face of such conflicting opinions, information is needed as to what happens to the victims of silicosis before they reach the stage of total disability for work.

We know that in the quarries of Vermont and Massachusetts there are men who have worked in stone all their lives, who are getting along with the vital capacity of their lungs reduced to a fraction of normal capacity. If you do not rush them, they can do a day's work. However, they have no reserves. But their disability is not reflected in their earning capacity, because their skill and experience offset satisfactorily to the employer their lack of physical speed. These men usually have a strong sense of family responsibility, and with great determination they go on with their accustomed work, often with undiminished earnings. Those who base their generalizations upon this segment of experience may argue that there is no such thing as partial disability from silicosis, so far as the workmen's compensation laws are

concerned, because there is no wage loss attributable to such disability. This contention comes more easily from a person who does not himself have silicosis, than from those who have to struggle for breath. In short, one segment of experience, as to silicosis, is commonly disposed of, in workmen's compensation administration, by the contention that there is no such thing as industrial disability, partial in degree, from uncomplicated silicosis. On the other hand, some spokesmen for organized labor insist that the prevailing practice of not compensating partial disability from silicosis is unjust, and some of them are becoming militant in respect to this grievance.

While some States do not provide compensation for partial disability for silicosis, some jurisdictions have had considerable experience with taking care of such cases in one way or another. The main needs are for maintenance, with or without medical aid, and for rehabilitation and for jobs. Under varying types of law, the maintenance payments may be on the basis of wage loss when the worker has to change to work that is less exacting physically. Or there may be payment based upon estimated "degrees" of nodulation. The rehabilitation provision may be with, or without, a special award to cover the roughly estimated cost to the worker of changing from one kind of employment to another. These varying provisions can, of course, either be used well or abused.

The rehabilitation type of provision, for the silicotic, at first glance looks ideal, especially if the attempt at change of employment is accompanied by a lump sum payment. However, it should be remembered that in Wisconsin the provision for a hardship payment is used by the administration to discourage employers from discharging their workers when an X-ray shows nodulation. Some types of rehabilitation provisions can be used primarily to close out the employer's future liability, rather than to improve the health and status of the worker which is the true purpose of rehabilitation. If there is an allergy, an effort should be made to arrange a satisfactory change of employment. However, the circumstance that a worker gets a new job, does not necessarily mean that he will either keep it or be able to get continuous employment at other jobs.

Where the shop housekeeping has been cleaned up and the dust removed from the air, it seems preferable not to interrupt the accustomed employment of a worker who has silicosis because, in many areas, when he has been thrown on the employment market it is very hard for him to get another job. In part because of the way some compensation provisions are written, in some States employers will not hire a man who has an unfavorable chest X-ray. Faulty compensation bill drafting, together with oppressive hiring practices, shunt too many truly employable persons toward the permanent total disability classification, or toward the public welfare rolls.

So far as New York is concerned, compensation for partial disability from silicosis is still a problem for the legislature. If compensation is provided by law for this category of disability, the New York Workmen's Compensation Board will of course face another disability rating problem. At this point, it should be said that in compensating occupational disease cases administration is hampered by the lack of early and correct diagnosis of disease. Inexpert diagnosis and the lack of wide experience with industrial processes, sometimes give a false start in the investigation of cases. Without competent and prompt diagnosis, maximum benefits do not flow from treatment. At present, so far as the workmen's compensation authority is concerned, the two stages of handling occupational disease cases are almost completely separated. The first stage is diagnosis and treatment; the second stage, disability rating and adjudication.

In many States today there is a hue and cry about the high cost of workmen's compensation. Of course, the public should be concerned about all excessive costs. However, in discussions of the cost of workmen's compensation the public is frequently misled into supposing that the legal benefit rates are too high or that the workmen's compensation administration is excessively liberal in its interpretations and awards. A properly worded statement would be that probably in all States the workmen's compensation costs are too high in relation to what is delivered in payments and services. What the public has not been told and does not now understand is that the major source of unnecessary loss is not

the administrative processing of claims -- which, of course, is not perfect -- but faulty diagnosis and treatment.

At the present stage of administrative development in the States, the workmen's compensation authority often has the sad task, when settling or adjudicating both disease and accidental injury cases, of mopping up the results of prolonged unsatisfactory medical care. How much longer this situation will be tolerated, especially by the more advanced administrations, remains to be seen.

I have mentioned two historical stages of development in the provision of occupational disease coverage in the States. In the first, the main preoccupation was that of shielding industry from allegedly unbearable cost. In the second the main preoccupation was on compensating, without discrimination, all victims of occupational disease. We are now in a third stage of historical development in workmen's compensation, with emphasis upon prompt and complete restoration of injured workers to their fullest work capacity. Because of an obsession with legal problems, many of them excessively technical, we have been late in waking up to the possibilities for reducing both human losses and monetary costs in the field of workmen's compensation, both through obtaining the right type of specialized medical care and by supplementary teamwork in medical care, which means what we have called "rehabilitation."

The advent of this new stage of development calls for an integration of the supervision of medical care which, of course, eventuates in disability rating and adjudication. At the present time administration deals chiefly with what is really only a fragment of the total medical problem. A report by the late Dr. Howard N. Prince on Medical Examining Facilities of the New York State Workmen's Compensation Board, several years ago, contains this statement: "The Chief Medical Examiner should institute an integrated medical program for workmen's compensation." Such a thing has not yet been done in any State. This failure is responsible, not alone for huge monetary losses, but also for tragic and unnecessary human loss. As you know, the monetary cost of workmen's compensation arises from, and is augmented or diminished

by, the extent of the human loss. We worry about monetary loss -- perhaps we ought to worry more about it -- but our paramount concern should be with the human loss. If we reduce the human loss, the monetary loss will dwindle also.

Our effort in New York is to protect injured workers to the fullest possible extent. If the programs of prevention and restoration are fully developed, the cost of adequate payments and services for injured workers should not be oppressive to industry. At present the program of prompt and full restoration of injured workers, in New York and elsewhere, is the most undeveloped segment of workmen's compensation administration. The resulting loss, both in human and monetary terms, may well be appalling. We shall study how best we can serve the injured worker and, as new light is thrown upon the task, will confidently count upon the cooperation of our people in perfecting the New York performance, so far as may be humanly possible.

Office of the Chairman
Workmen's Compensation Board
State Office Building
Albany, New York
Albany 3-5511, Ext. 333

For Release

Address of Hon. Mary Donlon, Chairman of the
New York State Workmen's Compensation Board,
at the Seventh Saranac Symposium at Saranac
Lake, New York, Friday, September 26, 1952

VIEWPOINTS ON COMPENSATION FOR PULMONARY AND
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It happened, however, that in one or two States where the original injury coverage provisions had been left broad and simple, omitting the restrictive word "accident", the courts held that the word "injury" included occupational diseases. Such States therefore started, almost by "accident", with the same compensation coverage of occupational diseases as of accidental injuries. Other States observed the result and liked it. The prevailing pattern in this country now is full or general coverage of occupational diseases. In this respect America leads the world. With almost every session of the legislatures, the column showing the list of States with schedule or restricted coverage of occupational disease shrinks, while simultaneously the column showing the list of full coverage States lengthens.

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on a lower level than for accidental injuries, either as to cash payments or medical care, or both. The laws in some States are still cluttered with discriminatory details that are hard to get rid of. Such petty and harassing restrictions are a liability to insurance carriers and employers because they arouse hostility in the ranks of labor, the consequences of which are out of all proportion to the pinch-penny savings effected by the restrictive provisions. This is beginning to be apparent in the conflict as to compensability of partial disability from silicosis. Such things keep workmen's compensation laws in politics and they should be above political considerations.

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When occupational disease problems are seen in human terms, appropriate action may be expected. Some of the difficulties of coverage may be obscure because the flood-light of research has not yet been turned upon them. On the other hand, some of the remaining tasks for legislatures can be approached through demonstration. In a certain agricultural State, there had been persistent and formidable opposition to occupational disease coverage. Committees had reported adversely on the proposal. There had been the usual negative arguments. It was insisted in the first place that there was no occupational disease problem

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In many States today there is a hue and cry about the high cost of workmen's compensation. Of course, the public should be concerned about all excessive costs. However, in discussions of the cost of workmen's compensation the public is frequently misled into supposing that the legal benefit rates are too high or that the workmen's compensation administration is excessively liberal in its interpretations and awards. A properly worded statement would be that probably in all States the workmen's compensation costs are too high in relation to what is delivered in payments and services. What the public has not been told and does not now understand is that the major source of unnecessary loss is not the administrative processing of claims -- which, of course, is not perfect -- but faulty diagnosis and treatment.

At the present stage of administrative development in the States, the workmen's compensation authority often has the sad task, when settling or adjudicating both disease and accidental injury cases, of mopping up the results of prolonged unsatisfactory medical care. How much longer this situation will be tolerated, especially by the more advanced administrations, remains to be seen.

I have mentioned two historical stages of development in the provision of occupational disease coverage in the States. In the first, the main preoccupation was that of shielding industry from allegedly unbearable cost. In the second the main preoccupation was on compensating, without discrimination, all victims of occupational disease. We are now in a third stage of historical development in workmen's compensation, with emphasis upon prompt and complete restoration of injured workers to their fullest work capacity. Because of an obsession with legal problems many of them excessively technical, we have been late in waking up to the possibilities for reducing both human losses and monetary costs in the field of workmen's compensation, both through obtaining the right type of specialized medical care and by supplementary teamwork in medical care, which means what we have called "rehabilitation."

The advent of this new stage of development calls for an integration of the supervision of medical care which, of course, eventuates in disability rating and adjudication. At the present time administration deals chiefly with what is really only a fragment of the total medical problem. A report by the late Dr. Howard N. Prince on Medical Examining Facilities of the New York State Workmen's Compensation Board, several years ago, contains this statement: "The Chief Medical Examiner should institute an integrated medical program for workmen's compensation." Such a thing has not yet been done in any State. This failure is responsible, not alone for huge monetary losses, but also for tragic and unnecessary human loss. As you know, the monetary cost of workmen's compensation arises from, and is augmented or diminished by, the extent of the human loss. We worry about monetary loss -- perhaps we ought to worry more about it -- but our paramount concern should be with the

human loss. If we reduce the human loss, the monetary loss will dwindle also..

Our effort in New York is to protect injured workers to the fullest possible extent. If the programs of prevention and restoration are fully developed, the cost of adequate payments and services for injured workers should not be oppressive to industry. At present the program of prompt and full restoration of injured workers, in New York and elsewhere, is the most undeveloped segment of workmen's compensation administration. The resulting loss, both in human and monetary terms, may well be appalling. We shall study how best we can serve the injured worker and, as new light is thrown upon the task, will confidently count upon the cooperation of our people in perfecting the New York performance, so far as may be humanly possible.

Chapter Thirty-one

The Functions and Value of Medical Boards and Medical Examiners
in Controverted Compensation Cases

Remarks by Lemuel C. McGee, M.D.

The foreword on the program.....

.....

.....in workmen's compensation.

OK
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man later on showed a nice cosmetic result. That is the point of view that I am presenting in connection with the problems of the medical board in workmen's compensation.

Thank you.

(Applause).

BY MISS DONLON:

Thank you, Doctor McGee, for that introduction to the discussion of functions and values of medical boards and medical examiners in controverted compensation cases and for that very interesting little example of what your company did outside its requirements to do so. I'm sure that those who, like Doctor McGee, approach these problems with a more human concern for people, would always try to do the extra little thing as nicely as he was able to do it in that case.

I think, of course, in a controverted compensation case such as we're discussing, our problem is, what are the issues under the compensation laws, and I'm sure Doctor McGee was only telling us that laws can not be written to cover every situation, and that you do have to bring a human viewpoint to it.

Now, as to the medical boards and medical examiners, New York, of course, has both medical examiners and in the dust or chest diseases, dust inhalation and chest diseases, a medical board of long standing, and I speak with pardonable pride, of the great ability in this field. We had hoped that all three members of that board would be with

Remarks by Edgar Mayer, M.D.

In the state of New York, prior to 1935, certain.....

.....

.....as boards, as time goes on.

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us today, but professional engagements have kept away from us, both Doctor Amberson and Doctor Whipple, whom I'm sure many of you know. However, we have the good luck to have the chairman of our board of chest consultants here in New York State, with us today.

I think anything I could say to people, as true to their experience in this field, about Edgar Mayer, would be telling you something that you already know. It has been one of my real privileges since I came into the field of workmen's compensation administration, to work closely with him in handling of problems that arise in the controverted cases under the different provisions of law, first our old Article 4a of limited coverage, and now the more general coverage that we have of the dust diseases in workmen's compensation.

He is well known here at Saranac where he has worked long. He known wherever society, learned societies meet to consider this problem. He is a very able physician in this field. I present to you the chairman of the New York State Board of Chest Consultants, Doctor Edgar Mayer.

BY DOCTOR MAYER:

I want to apologize for Doctors ^{*the absence of*} Amberson and ~~Doctor~~
Whipple. Unfortunately, ^{*with*} ~~as far as~~ Doctor Amberson goes, you
~~probably know that~~ the College of Surgeons is meeting in
New York, and Bellevue has to take its part in the proceedings.

It's very obvious that in the past five years, there have been very outstanding advanced contributions made here in this field by the excellent group that are working in Saranac and to whom we're all so much indebted.

how we function and if I may say that I will, rather immodestly say what I consider as our values.

I yesterday was approached by one of my legal friends, an advisor to industry here who said, Amberson and Whipple aren't here and you're going to face us alone? And I do do that with much trepidation, but I should probably feel that some representative of labor who would be here would make the same remark to me.

original paper begins here?
 In New York State, prior to 1935, certain occupational diseases were compensated in accordance with a schedule. Silicosis and other dust diseases were not included in this schedule. The laws of 1935, Chapter 250, or 254, extended the Workmen's Compensation coverage to all occupational diseases and silicosis then became compensable in New York State.

Because of the lack of knowledge of the extent of the accrued liability, the premium rates for this insurance were considered high. In 1936, the law was amended -- ~~1~~
You can
 wish you'd look these up -- Chapter 887, limiting the coverage and compensation benefits, providing for dust control and prevention of silicosis and other dust diseases, and providing for the establishment of a committee of three expert consultants to study each claimant and to inform the Industrial Commissioner and the Industrial Board of their opinion as to the findings in such cases.

The law was again amended in 1947 to provide full benefits for disabling silicosis and other dust diseases under Chapter 431 and at the present time, the law that deals with the work of the expert consultants if - is found in Paragraph 49-a of the Workmen's Compensation Law.

Now, as to our functioning, as soon as a claim for silicosis compensation is filed with the board, it is referred to the after care service of the Workmen's Compensation Board. This after care service obtains a chronological history of the claimant's illness, and a detailed chronology of his employment record. The claim is brought to the attention of the employer and the insurance carrier and the claimant is referred, for examination, to a physician designated by one of the latter. A hearing is held in the case before a Referee in compensation, and if a claim is controverted by either or both sides, the patient is then referred to a member of the board of expert consultants.

The examination requires the taking of very careful occupational history from the beginning of the patient's industrial life, including all aspects of exposure to dust, and it is at this time that the file is referred to the Division of Industrial Hygiene of the Labor Department for an examination of the working condition surrounding the claimant and for such dust diseases as may be necessary in order to evaluate for such disturbance as may be necessary in order

to evaluate the dust hazard.

In certain cases, the Division of Industrial Hygiene summarizes additional data and opinions with reference to past exposures suffered by the claimant. The examination also includes a very complete physical examination, a fluoroscopic chest study, and (we carry out the procedure that has been worked out here) diaphragmatic rise with expiratory timing, as an aid in evaluation of emphysema, as well as inspiratory and expiratory x-rays. Occasionally we take lateral and oblique x-rays, and on rarer occasion planigrams.

Often we take electrocardiograms, especially in vascular hypertensives, and laboratory studies are made as are necessary in the opinion of the examiner, such as repeated sputum studies or even gastrics, blood examinations, erythrocyte sedimentation rates, and so forth.

In evaluating the hazards of occupation exposure,

the examiner is aided by the report of the Division of Industrial Hygiene which has made (or is now requested to be made) spot studies of the plants and the working conditions.

In evaluating disability, the examiner may call for special physiological studies of pulmonary function with the best available methods. When exact diagnosis in evaluation of disability is impossible through one examination, we are allowed (as Doctor Merewether has mentioned is the custom in England) to make follow-up and auxiliary studies.

On occasion we may classify a claimant as temporarily totally disabled when, for instance, it is impossible to evaluate the activity of a confluent shadow in the lung. We may classify such a patient as totally temporarily disabled and re-evaluate his condition at the end of three months. On rare occasions hospitalization is carried out when additional studies are necessary to establish diagnosis through bronchography, bronchoscopy, angiography or surgical biopsy.

here of whether we're dealing with beryllium; there was a very questionable exposure to beryllium, or whether we're dealing with a big sarcoid or whether we're dealing with some other type of unexplained fibrosis, a condition which we can not define without a surgical biopsy.

If disability may be related to allergies ^{and} in industry through exposure to ^{certain} various dusts, we are allowed a consultation opinion of ^{an allergist} ~~analogs~~ and this is often recommended. If pulmonary disease may be related to the upper respiratory tract, special examination by a nose and throat specialist is requested.

Finally, the completed history and the physical examination with the conclusions as to diagnosis and causal relationship and degree of disability is reported. Copies of the reports are sent to all interested parties, as well as to the attending physicians and to the medical director of any hospital or sanatorium in which treatment has been carried out. If the claim is then controverted, the case is referred back for examination to the complete board.

The contents of such report of the committee, introduced in evidence, constitutes prima facie evidence of fact as to the matter contained therein and any of the material of such report is then subject to examination upon demand.

In the event of a claim for death benefits, one

member of the board examines all available evidence pertaining to such claim, including the medical and the hospital records, the x-rays and other reports that are made during the lifetime of the deceased, as well as any autopsy findings and shall render the findings and report thereon.

Now, as to the values of the board, I am putting this very briefly. I think Doctor McGee presented the function of such medical boards very well. Our board, impartial in nature, has proved of great value from both the medical and legal aspects. We are free of any obligation except that of impartiality to both employer and to claimant. We can freely acknowledge a conclusion to be one based upon opinion from experience only, when medical evidence does not permit scientific proof. We are not necessarily restricted to one examination to establish diagnosis, causal relationship and disability. Frequently conclusive diagnosis can not be made, as you well recognize, until time has elapsed for the development of significant subjective and objective signs.

Examinations of all three of us in hearings before referees are frequent and are characterized by unrestricted and frank questioning by counsel of both claimant and employer, as well as by the referee.

We think that the impartial character of our conclusions, both in our reports as well as in our testimony,

have served to gain the board the necessary respect of the referees, as well as that of the counsel on both sides.

From the medical point of view, we have attempted to set a high standard for thorough and conscientious examination in reaching a diagnosis and establishing causal relationship in evaluation of disability.

Our work, in part, has served to highlight many of the different clinical problems of industrial pulmonary diseases, as well as the problems in evaluating the degree of disability in pulmonary disease, and accordingly, we were among the very early ones to acknowledge this and to call freely upon the aid of the physiological laboratory.

We believe that such openmindedness in helping to solve these difficult clinical problems in industry, may ultimately do much to define, with the cooperation of the workers here in Saranac Lake (with whom we have been fortunate to be in such intimate contact), ~~we will ultimately, be better able to define, perhaps,~~ what can be expected in the future by way of aid from the laboratory.

During this week, I have been impressed with some of the difficult future problems that ^{may} ~~will probably~~ arise because of the added information that our British friends have brought us, as well as the information that has come from Doctor Verwald and Doctor Wright's laboratories.

~~Number one~~, I can see where our legal friends will be asking

us to define the word 'pneumoconiosis' which, as you have learned at this session, is now going to be re-defined. I can see where new problems will come up in relation to what were formerly considered as inert dusts. You saw in the excellent pathological sections of Doctors Vorwald and Fletcher focal emphysema, which could, in certain instances, eventually lead to total disability and which may be caused by a sufficient quantitative deposit of so-called inert dusts.

In relation to graphite and carborundum dust -- dusts that we have been regularly considering inert -- should we now accept the suggestion that there are no such inert dusts when they are inhaled in excessive amounts and so seen radiologically?

This may become a problem in the evaluation of disability. In the future we'll probably be calling more frequently upon our Division of Industrial Hygiene for evaluation of the quantity of such dusts.

Doctor George Wright, with Doctor Leonard Bristol, in their presentation of emphysema as an independent disturbance

of the lung, separate from that which is derived from industry, have placed before us a problem that has disturbed us for many years. We recognize that much emphysema is of the so-called essential type unrelated to industry and presents disturbing problems to us in defining a causal relationship to industrial exposures.

The problem of cancer of the lung in relation to industry also presents itself. Do we have now to give serious consideration to the chromates and to asbestos (I think all the other exposures have been rather well defined) as causative factors to industry? The Germans, as you have heard this week, have granted causal relationship to asbestos in the development of cancer of the lung.

These are some of the problems that I can see will confront us more often in the future and

on which we will have to have much more accurate information. (~~Thank you~~).

on which we will have to have much more accurate information as time goes on. Thank you. (Applause).

BY MISS DONLON:

Thank you, Doctor Mayer. I can only say for my colleagues who are administrators of workmen's compensation from other states, and I see several of them here in the audience, that I wish you the great good fortune of being able to have a medical board chaired by so distinguished a person in this field as Doctor Edgar Mayer, but don't any of you bid for him, because we want him here in New York.

Now, to conclude this part of the discussion, and then we will have it followed by a period for discussion from the floor, we have, of course, the legal viewpoint. We lawyers always have to come in on this partly, and so we are going to have the legal viewpoint, and we have a gentleman to discuss it, a lawyer who is very well qualified to do so. He is the Assistant Counsel of Republic Steel Company at the home office in Cleveland, Ohio, but we here in New York know him and have worked with him, and I'm sure that many of you from other states also knew and have worked with him. Without anything more, I turn the floor over to him, Mr. A. J. Gentholtz.

BY MR. GENTHOLTS:

(Mr. Gentholtz read a prepared paper which is on file at the Saranac Laboratory).

BY MISS DONLON:

Remarks by A. J. Gentholt

At the outset.....

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.....of Workmen's Compensation Laws.

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THE FUNCTIONS AND VALUE OF MEDICAL BOARDS AND
MEDICAL EXAMINERS IN CONTROVERTED COMPENSATION CASES

At the outset, I am compelled to state that it seems rather ridiculous for me to attempt to either discuss this subject with the members of this panel or to present any sort of a formal paper about it to this audience, for I fully realize how authoritatively anyone of you can discuss it.

I think it would be helpful to report briefly as to how far legislation establishing Medical Boards of Review has progressed in the United States. The establishment of Medical Boards of Review was not thought of until the enactment of occupational disease laws. Today there are only five states that do not have some type of occupational disease coverage; namely, Kansas, Louisiana, Mississippi, Oklahoma and Wyoming. By the introduction of this type of Workmen's Compensation claims founded on disease arising in industry, the legislatures in many states have enacted laws, in addition to occupational disease amendments, establishing Medical Boards of Review.

The picture is somewhat different, however, when we consider the legislative action which has taken place in the establishment of Medical Boards of Review. Although occupational disease has been recognized as a proper administrative function under Workmen's Compensation Laws in 43 states, only eighteen states have enacted laws establishing Medical

Boards of Review of one kind or another. The states are:

| | |
|-------------|----------------|
| Arizona | N. Carolina |
| Colorado | Ohio |
| Georgia | Oregon |
| Idaho | * Pennsylvania |
| Iowa | S. Carolina |
| Maryland | S. Dakota |
| * Michigan | Texas |
| * Minnesota | Utah |
| Nevada | W. Virginia |

* The Minnesota and Michigan statutes were held unconstitutional in 1945 and 1946 respectively; by court decision in Pennsylvania the statute became inoperative in 1946.

New York State has in a manner approached this type of legislation by the adoption of a statutory amendment (Section 13, sub-section (d) of the Compensation Act) providing for the appointment of an impartial specialist in injury cases - the report and findings of such specialist not being binding on anyone. If this system is to prevail it would seem proper that both claimant and the employer should be given the absolute right to obtain an examination by a disinterested specialist.

It is interesting to observe that of the 18 states enacting laws establishing Medical Boards of Review, only 5 states (Georgia, Idaho, Maryland, Minnesota and Ohio) have established such Boards with final jurisdiction as to medical facts.

In addition to the Medical Boards of Review in the states which have been enumerated, you of course are familiar with the procedure which has been adopted in the establish-

ment of tribunals characterized as "Silicosis Boards of Referees". In many instances these tribunals are separate and distinct from the Medical Boards of Review. The Silicosis Boards, as the name implies, are restricted only to the consideration of silicosis cases or other respiratory diseases. The Medical Boards of Review, on the other hand, are established for the consideration of medical evidence pertaining to occupational disease cases generally. In some states the right of appeal is available from the decision of the Silicosis Board of Referees to the Medical Board.

The states which have enacted laws establishing Silicosis Boards of Referees are as follows:

| | |
|---------------|----------------|
| Arizona | Nevada |
| Arkansas | N. Carolina |
| Colorado | Ohio |
| Georgia | Oregon |
| Idaho | * Pennsylvania |
| Iowa | S. Carolina |
| Maryland | S. Dakota |
| * Michigan | Texas |
| * Minnesota | Utah |
| West Virginia | |

* The Minnesota and Michigan statutes were held unconstitutional in 1945 and 1946 respectively; by court decision in Pennsylvania the statute became inoperative in 1946.

In New York State the Compensation Act has been amended to provide for the appointment of a "committee of expert consultants on chest diseases", three in number, which also provides for an examination by one of the members thereof. There is nothing in the statutory law which makes the opinion or report of the experts conclusive or binding on anyone.

Of the 19 states which enacted laws establishing Silicosis Boards of Referees, only 5 states (Georgia, Idaho, Maryland, Minnesota and Ohio) established such Boards with conclusive and final jurisdiction.

The experience in Minnesota, Michigan and Pennsylvania should be of interest since in these three states Medical Boards of Review were established by legislative action but today are inoperative because of court decree.

The Michigan Workmen's Compensation Act was amended, effective October 29, 1937, by incorporating the Occupational Disease provisions of the Law, better known as Part VII of Act No. 61. Briefly stated, this amendment, in addition to providing Workmen's Compensation awards for Occupational Disease claims, authorized the appointment by the Workmen's Compensation Board, or any member thereof, of

"a commission of three qualified impartial physicians to examine the injured employee and to report. The report, when signed by at least two of the members of said commission, shall be final and conclusive as to the condition of said employee with respect to the alleged disease or diseases. . . ."

Following such legislative enactment, a number of cases were prosecuted through the court, culminating in the finding by the Supreme Court of Michigan in the Dation v. Ford Motor Company case, 314 Mich. 152, 22 N.W. (2nd) 252, decided in 1946, that such law

"is unconstitutional as denying due process, since the Legislature cannot circumscribe judicial power of the courts by making the

factual findings of the agencies conclusive, even though the findings are wrong. 'Due Process of Law' requires notice and opportunity to be heard, and it imports the right to a fair trial of the issues involved in the controversy and a determination of disputed questions of fact on the basis of evidence. . ."

Since the abolishment of the Medical Board of Review by court decree in 1946, the administration of controverted occupational disease claims in Michigan has been prosecuted in the same manner as injury cases. A quick survey of the situation existing today in Michigan indicates that representatives of employer and claimant alike prefer the existing administrative procedure rather than the methods which were required to be followed under the 1937 act, which in my opinion, established an imperfect setup under the Medical Board of Review principle.

Similarly, in Minnesota the Medical Commission (as the Medical Board of Review was called in that state) only existed for a very short period of time. In Minnesota the Workmen's Compensation Act of 1913 was amended in 1943 to include occupational disease provisions. At the same time a Medical Commission (or so-called Medical Board of Review) was established. The Medical Commission under the statute consisted of three doctors selected from a panel of 15, chosen by the Dean of the College of Medicine of the University of Minnesota, the Council of the Minnesota State Medical Association, and the Governor.

The findings and conclusions of such Board were adopted by the Industrial Commission in so far as medical questions were concerned and such decisions were final. This system lasted for a period of approximately two years until the statute was held unconstitutional in the case of Hunter v. Zenith Dredge Company on July 6, 1945. The basis for the decision was the same as in Michigan in that the Medical Board of Review statute was found to violate the due process provisions of both the Minnesota and Federal constitutions.

Since the abolishment of the Medical Board of Review in this state, the procedure has reverted to that formerly in existence whereby under the statute, the Industrial Commission is authorized to appoint one neutral physician whose findings and conclusions shall be furnished all interested parties; either party may within five days after receipt of such report demand cross-examination of the neutral physician. The report and cross-examination are both considered as evidence.

A somewhat similar situation arose in Pennsylvania, following the enactment of an amendment to the Compensation Law on July 2, 1937, which made certain specified occupational diseases compensable. At the same time the Workmen's Compensation Board, or Referee, was authorized to appoint a Medical Advisory Board in cases involving claims for silicosis, the Board to be composed of one or more duly qualified,

impartial physicians or surgeons to be selected from a panel made up of names from lists of doctors furnished by the deans of all of the legally recognized medical schools in Pennsylvania. The so-called "Medical Board" in this instance was authorized to conduct clinical, pathological, and x-ray examinations, and to file with the Workmen's Compensation Board or Referee its written report stating further its opinion as to whether or not the claimant suffered from silicosis or anthraco-silicosis and whether such disease resulted in any disability and, if so, as to what extent.

The Workmen's Compensation Board or Referee was authorized to consider such so-called Medical Board report only as evidence of findings and to be considered with any other medical evidence offered on behalf of the claimant or the employer; the doctor or doctors serving on such Medical Board were obligated to appear and be subject to examination and cross-examination upon written request.

In 1941 this Act was amended again by authorizing the Governor of the state to request the deans of all of the medical schools in Pennsylvania to serve as a committee in the nomination of 10 physicians especially qualified for membership on the Medical Board; three to be roentgenologists and three to be pathologists, these names to be certified to the Secretary of Labor and Industry. From this list of names, with the approval of the Governor, the Secretary of Labor was to select the Medical Board consisting of three

members, including one roentgenologist and one pathologist. Under this amendment, the duties and powers of the Board were not materially different than under the original 1937 Act.

It is interesting to note that no Medical Board was ever constituted or appointed in Pennsylvania until May 1950. Following the appointment of the Medical Board in 1950, a number of cases were prosecuted under such administrative procedure. All of these claims were occupational disease cases of alleged silicosis or anthraco-silicosis. In every instance the Medical Board, without hearing and simply from examination of the claimant, and apparently without study of the record, made what purported to be final findings of fact without reporting their findings to the Workmen's Compensation Board. Upon appeal of all of these cases to the Common Pleas Court of Pennsylvania, the court held in each instance that the Board had exceeded its authority.

Apparently, the unsatisfactory method pursued by the Pennsylvania Medical Advisory Board caused Pennsylvania Legislature in January 1952 to repeal sections of the Occupational Disease Act, completely abolishing the Medical Advisory Board.

Because of the failure in operation of Medical Boards of Review in three great industrial states in this country, one might question whether the principle of this sort of medical tribunal in the administration of controverted compensation cases is proper.

From my own point of view, I do not believe that this is so and in support of such position, it is appropriate to point to the experience of Ohio where the adoption and use of both Medical Boards of Review as well as Silicosis Referees have proven quite satisfactory.

The first Occupational Disease law was passed in Ohio in 1921 covering four specific occupational diseases. In 1924 an appropriate constitutional amendment was passed encompassing all of the occupational disease provisions of the Workmen's Compensation Law. At the present time, following several amendments to the Occupational Disease Law, it is considered as a blanket statute permitting awards in all occupational disease cases, in addition to 24 diseases specifically enumerated.

In 1937 the Ohio Legislature enacted a Medical Board of Review statute. At the same time an amendment was enacted establishing a Silicosis Board of Referees. Under the Ohio Act, approximately 500 recognized physicians and surgeons are selected as a panel. From this panel individual members of Boards of Review are appointed from time to time. These physicians and surgeons reside and practice all over the state. In addition, some of these appointees are residents outside of Ohio as the law does not restrict such appointments to residents of Ohio. The selections are made by the Dean of Medicine of Ohio State University, the Director of the State Department of Health, and the Head of the Department of Medicine of the Industrial Commission.

From time to time, as Medical Boards of Review are required to decide controverted occupational disease cases, the three appointive agencies select from the panel of approximately 500, three physicians for each meeting of such Medical Board of Review. The same physicians are rarely called upon the second time, for by reason of their professional duties and practice they may be unavailable for such service. The list has remained more or less permanent except for resignations or removal because of death, illness or other personal reasons.

The administration of controverted occupational disease claims under this setup is as follows:

The controverted claim initially is processed through the Medical Department of the Commission for the recommendation of the Medical Department for allowance or disallowance, by the Industrial Commission itself or by one of the several Claims Boards which operate in various sections of the state.

Upon disallowance of the claim by either the Claims Board or the Industrial Commission itself, the claimant has the right of appeal to the Medical Board of Review. Upon appeal the entire file, including all medical proof, is available to the Board. The complete record in the case is read and studied by the Medical Board of Review in the presence of the claimant, his counsel and employer's representative. The claimant may offer additional medical proof and factual proof at such hearing. The Board may examine

claimant or refer him to a recognized specialist for further examination.

Now, here is the important difference in the Ohio Act as distinguished from many of the states which have enacted the so-called "Medical Board of Review" provision. If the decision of the Board is in favor of the claimant, such findings shall be binding upon the Industrial Commission. In other words, the decision is final and there is no appeal by the employer either to the Industrial Commission or to the courts.

On the other hand, should the decision of the Medical Board of Review be adverse to the claimant's position, an appeal may be made to the Industrial Commission. Experience, however, shows that this situation occurs very infrequently. There is no appeal to the courts on occupational disease cases, either by the claimant or the employer.

The Silicosis Board of Referees is appointed by the Industrial Commission and is generally recognized as being made up of men of outstanding qualification and training in diagnosis and treatment of respiratory diseases. The statute is specific in Ohio that all claims for silicosis, whether controverted or not, must be referred to the Silicosis Board of Referees for examination and recommendation as to diagnosis, extent of disability and other medical questions connected with the claim. Such Board has the authority to employ other specialists, roentgenologists or pathologists in case of death, in the administration of this work.

The procedure with respect to the administration of silicosis claims is as follows:

The claim is first investigated by the Industrial Commission to determine whether the statutory provisions of the law have been complied with; such as (a) exposure; (b) disability within the statutory period; (c) diagnosis of one or more licensed physicians.

A synopsis of the case is then prepared for the use of the Silicosis Board of Referees. In every instance where the claimant is physically able, an independent medical examination is conducted by such Board at the time of hearing. If the claimant is physically unable to be present, all available medical data is obtained from the hospital where the individual is receiving treatment.

Upon completion of such study and examination by the Board, the claim is heard in Columbus, none of the interested parties being present. The findings of the Board are then referred to the Industrial Commission, who fixes a time and place of hearing upon the report of the Silicosis Board of Referees. Copies of the findings and conclusions of the Silicosis Board of Referees are furnished all interested parties. An adverse finding by the Silicosis Board may be appealed to the Medical Board of Review, whose decision shall be final, the same as in occupational disease cases generally. If an award is made, the employer may file an Application for Reconsideration before the Industrial Commission; again this

decision is final with no appeal.

Under this form of administrative procedure by such Medical Boards of Review, as well as Silicosis Boards of Referees, statistical information shows that approximately 50% of the silicosis claims heard are allowed. Approximately 30% of those disallowed are appealed to the Board of Review; of those which are appealed, approximately 10% are allowed.

As to Occupational Disease claims generally, statistical information shows results quite similar in experience to silicosis claims. Approximately 90% of the cases heard are allowed. Of the 10% disallowed, 90% are appealed to the Industrial Commission; of those which are appealed, 10% are allowed.

Thus it will be observed that in the administration of occupational disease claims in Ohio, the experience seems to clearly show that the functions of such Medical Boards, as well as Silicosis Boards of Referees, have proven to be a distinct advancement in the administrative procedure in controverted compensation cases. Experience in Ohio has shown that among representatives of Industry and Labor, lawyers and physicians, the present procedure, while not entirely satisfactory in some respects, functions in such a manner that little or no resistance or criticism is heard respecting it.

The proof of such a statement is found in the fact that since the advent of these Medical Boards of Review,

beginning in 1937, in only five instances has an attempt been made to set aside the decisions of such Boards by appeal in mandamus to the Supreme Court of Ohio. In each of these instances the Court found either originally or on appeal to the Supreme Court of the United States, that the decision below was sound, that there had been no denial of the due process of law and that the position of the Medical Board of Review should be sustained.

To summarize, therefore, as to the essential reasons for the apparent satisfactory administration procedure of Medical Boards and Silicosis Boards of Referees, I think they can be stated as follows:

1. General recognition of complete impartiality in selection of highly qualified physicians.
2. The initial investigatory procedure prior to the claim being presented to such Boards to determine compliance with statutory provisions of law, such as exposure, disability within the statutory period, preliminary diagnosis of one or more licensed physicians, and period of employment within the state.
3. Exclusive jurisdiction as to diagnosis with no appeal to the Industrial Commission or to the courts.

I believe it would be helpful here to paraphrase the subject assigned by stating it this way - "What can be gained by having Medical Boards of Review?" I realize full well in discussing this subject there are wide differences of opinion respecting it. You are all well aware of the divergent viewpoints existing among members of the legal profession

specializing in Workmen's Compensation claims representing claimants as well as those attorneys representing casualty insurance companies and large and small self-insuring employers. A similar difference of opinion, I believe, exists in the medical profession among many recognized top physicians and surgeons who are active in their practice in the administration of Workmen's Compensation.

Why does this condition prevail? I think one answer is that there are always deep-seated fears present among lawyers representing both claimants as well as the employer, that by the establishment of a Medical Board of Review, certain fundamental rights and privileges are taken away, thereby preventing either the injured person on the one hand, or the employer on the other, from having his day in court.

I am sure you have heard the statement made many times in discussions respecting this type of Workmen's Compensation administrative procedure, that no doctor, even though he has been appointed on a Medical Board of Review established by law, should be permitted to express an opinion without being subject to cross-examination. In my opinion, if a medical board is impartially and properly established there is no reason or necessity for the right of cross-examination to exist.

I am firmly convinced that in the enactment of statutes of this character, the Medical Board of Review can only function properly when it is constituted in such a manner

that it is completely impartial and removed entirely from any political or any other kind of influence. By that I mean that such Board should have complete freedom to decide cases independent of any pressure or influence from claimant, employer, labor representative, or anyone interested whatever in such claim. Further, the method of selection of the panel representatives for appointment on the Medical Board of Review should be controlled to assure selection of physicians and surgeons recognized generally by the public, the legal profession, industry, and the medical profession, as outstanding in the medical fraternity, not only from a professional but also from an ethical and moral point of view. Furthermore, when such Medical Board has been established by law in the manner thus described, then there is no prohibition against giving such Board final jurisdiction, when its decision is in favor of the claimant.

I therefore feel that, as heretofore stated, the establishment of properly constituted Medical Boards of Review in the administration of controverted compensation cases, can be found to be a distinct advantage in the administration of Workmen's Compensation laws.

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Smith v. Wilson Fdry. Co., 296 Mich. 484, 296 N.W. 654 (1941)
Walker v. Loselle Co., 305 Mich. 121, 9 N.W. (2nd) (1943)
Dation v. Ford Mtr. Co., 314 Mich. 152, 22 N.W. (2nd) 252 (1946)
Hunter v. Zenith Co., 220 Minn. 318, 19 N.W. (2nd) 795
Erickson v. Knutson, 54 N.W. (2nd) 118 (Minn.)

Wright v. Lehigh Valley Coal Co., 38 Pa. Dept. Rep. 13
 Dvorsky v. Lehigh Valley Coal Co., 38 Pa. Dept. Rep. 19
 Matylewicz v. Hudson Coal Co., 38 Pa. Dept. Rep. 1
 Unora v. Glen Alden Coal Co., 38 Pa. Dept. Rep. 29
 Pa. Legis. 1/2/52, Act No. 482, Sec. 4
 Plaughter v. Am. Viscose Corp., 147 Sup. Court 372
 Mandamus Acts of June 8, 1893, P.L. 345; Apr. 18, 1899, P.L. 84
 Mar. 19, 1903, P.L. 32; June 19, 1913, P.L. 526; May 13, 1925,
 P.L. 664; 12 PS Sec. 1911; Appeal of Hartranft et al., 85 Pa.
 433
 Bevis v. American Rolling Mill Co., 151 OS 293
 Jonak v. Beall, 136 OS 213
 Efford Case, 151 OS 109
 Venys Case, 153 OS 238
 Shovel Co. Case, 153 OS 451
 Bessler Case, 157 OS 297
 "Medical Proof in Workmen's Compensation Cases" Ohio Bar 22
 June 13, 1949, 169-74
 "Ohio Procedure in Occupational Disease" Ohio Bar 16, 5-24-43
 Ohio Bar 15, June 22, 1942
 New York Legislative Document (1932) No. 83
 New York Times, Dec. 31, 1933, Sec. 1, p. 12
 N.Y. Legislative Documents: (1920) No. 26; (1927) No. 69
 (1929) No. 49; (1934) No. 50 (u).
 Clinics, Vol. I, No. 6, April 1943, p. 1357
 Downey, E. H. Workmen's Compensation, MacMillan, N.Y. 1924, p. 71
 Journal of Am. Medical Assn., Vol. 121, No. 11, Mar. 13, 1943
 New York Academy of Medicine Bulletin, Vol. 17, 1941
 New York State Journal of Medicine, 1944
 International Assn. of Industrial Accident Boards & Commissions,
 U.S. Dept. of Labor, Div. of Labor Standards, Bulletins No.
 17 (1938), No. 53 (1942), No. 87 (1947), No. 119 (1949),
 No. 142 (1951)
 Hand, Learned, "Historical" (1901), 15 Harvard Law Review, 40,
 p. 56
 1947 Cumulative Supplement, Annotated Code, Md., Article 101-40
 Fraga v. Commissioner, 125 W. Va. 107, 23 S.E.(2nd) 641.

THE FUNCTIONS AND VALUE OF MEDICAL BOARDS AND MEDICAL EXAMINERS IN CONTROVERTED COMPENSATION CASES

The foreword on the program for this Symposium gives as the objective for the meeting "that industrial health be improved". What relation to that objective has the function of a medical board and medical examiners in controverted compensation cases?

It is agreed by many of us that the most useful result of laws granting compensation for industrial injuries is that of safer working conditions in industry. Will statutes covering compensation for occupational disease give support to the industrial hygiene movement similar to that which the statutes for injury gave to the safety movement? There is reason to believe that such a salubrious sequel may be had from the wise administration of laws dealing with occupational disease.

To this end it must be remembered that the laws refer to occupational diseases and not to the legion of non-occupational ills to which the human flesh is heir. By providing compensation for occupational diseases, accurate diagnosis and the recognition of correct etiology have been given new significance. If industry is penalized by payments for non-occupational afflictions there ^{and indust. hygiene programs to avoid} would appear to be less incentive for in-plant health programs as a responsibility of industry. I know of no state law that says the employer should pay for all illness contracted during the course of employment, including those of non-occupational origin. *occupational disease*

In these comments, I start with the premises (1) that compensation laws for both injury and disease arising out of and because of occupation are desirable in principle; (2) that good administration, even of inadequate laws, can reach equitable and worthy ends; and (3) that it is accepted as desirable to reduce errors in decisions to a minimum.

Competent medical diagnostic service from a team of medical examiners can aid in minimizing errors in compensation decisions. The past few years have seen a trend toward liberalization of benefits and full coverage of occupational diseases instead of schedule coverage. Forty-three states, in addition to Alaska, Hawaii, Puerto Rico, the District of Columbia and federal jurisdictions under the Longshoremen's Act, now have some form of compensation for occupational disease¹. In twenty or more states there is provision for medical examiners to furnish some type of service to the administrative set-up.²

Various reasons have been presented for a board of medical examiners. Some of these are the following:

(1) Controversy in compensation hearings usually arises from the effort to answer one or more of these questions³: Is there a disability? What is its degree? What is its cause? These are medical questions of fact and not of law. Their answer requires medical study and diagnosis.

(2) In industrial medicine, as in all aspects of medicine, the individual is paramount. To safeguard the interests of the injured or ill worker the physician must have the opportunity to investigate, to examine and advise as an expert. It is important that "there be a place for a quiet and objective discussion to dispel discrepancies or, at least, weaken them. Such consideration should be carried out in good faith with no end other than to serve the truth."⁴

(3) An understanding of medical terminology may best be given by a medical board. Disagreements, confusion and errors are oftentimes the result of simple problems of semantics. "The language of the craftsman must be made intelligible to those untutored in the craft."

(4) There has been agitation for some plan which will avoid the undesirable spectacle of incompetent and prejudiced testimony of medical witnesses selected for opposing litigants.

(5) A competent medical board can do much in selecting those patients who need special rehabilitation measures. Maximum limits on medical benefits are said to exist in the statutes of seventeen states.⁵ The worker needs to be made ready for the job before these benefits expire. Rehabilitation properly has become an important part of the compensation program. It is wholly logical to use the curative and retraining periods, following injury or disease, to restore and improve the earning power of the handicapped worker. Here is a place for guidance from capable medical examiners.

(6) "The fairness of the method of assessing the cost of workmen's illness against business as such, on the ground of aggravation of a pre-existing condition is coming more and more into question."⁶ Numerous degenerative diseases afflict the human body and go undetected for years. Progress toward serious disablement may be undetected by the victim, his co-workers, his family and even by his family physician. Medical examiners are our best recourse in dealing with the question of aggravation or acceleration of pre-existing disease and judiciously allowing the benefit of the doubt to the claimant worker.

(7) From time to time one observes the injudicious procedure of assuming that disease recognized following an accident was therefore due to the accident (*post hoc ergo propter hoc!*). A judge has been quoted as saying "Whether a particular event was an industrial accident is to be determined not by legal definitions but by the common sense viewpoint of the average man." I must say flatly that the "common sense viewpoint" has led repeatedly to egregious errors in medical diagnosis and that the "average man" has been of astoundingly little help in medical progress. Recently a woman bruised her breast, which event directed her attention to a tumor underlying the superficial injury. Common sense incorrectly led her to assume that the bruise caused the tumor. The tumor was a cancer antedating the bruise by at least a few weeks. She has probably been saved from death by cancer through early surgical

treatment which followed her good fortune in getting a bruise at that particular time.

The common sense viewpoint of the average man long insisted that the earth was flat. It was the uncommon wisdom of the few exceptional men who demonstrated that the earth was not flat. I do not wish to labor the point. Medical boards will make mistakes, but there will be fewer such mistakes than if medical decisions are made by those untrained in that field. If given the opportunity as technicians, competent medical examiners can reach closer to the truth based on the knowledge available to our generation than can lay administrators working alone.

(8) A medical board is a unique and highly useful center for the accumulation of new knowledge of disease processes and of their relation to occupation. Careful study of the accumulated experience of medical advisory boards with reports to medical journals will further clarify obscure disease and, in turn, facilitate more equitable administration of compensation benefits. An example of an informative and valuable contribution to knowledge is seen in a report by Watkins⁷, based on experience of the Medical Advisory Board of the Industrial Commission of Arizona.

It is generally agreed that medical testimony is indispensable when the existence of a disability, its degree and its cause are in doubt. The uncertainties inherent in occupational disease claims increase the need for medical guidance in compensation administration. Such guidance is particularly important in states where occupational diseases are covered under broad provisions, for questionable claims are more likely to be brought under such coverage and contexts based on causal relationships are more likely to arise.

The mechanism whereby the physician's maximal contribution can be made to the administration of the compensation system, I believe to be a legal matter. At the Saranac Symposium in 1947 I questioned the wisdom of any plan which

transferred legal responsibilities of a compensation commission to a physician or a medical board.⁸ It is repugnant to the Anglo-American traditions of justice to place a litigant at the mercy of witnesses he cannot see or challenge; to have his rights stand or fall on the basis of unrevealed facts which might be refuted or explained. Possibly a representative of the medical board should be available for questioning and cross-examining at the hearing. It may be desirable that the medical board review testimony from the hearing to see if there are additions to the information which was available to them when making their decision on medical facts.

Waters⁹ has said "From my own experience, I believe that medical boards and consultants appointed under the provisions of occupational disease statutes have materially assisted in the administration of the law. What agency is better qualified to pass on controversial medical questions than a trained, impartial medical board, thoroughly experienced in the subject matter of the disease for which compensation is sought?"

At a Harvard Law School dinner in 1895 the late Mr. Justice Holmes remarked, "An ideal system of law would draw its postulates and its legislative justification from science."¹⁰

The aim of social laws is justice and not abuse.

BIBLIOGRAPHY

1. Kalmykov, Andrew. Current Legislative Developments in Workmen's Compensation, address before Legal Conference, Annual Meeting, Industrial Hygiene Foundation, Pittsburgh, November 14, 1951
2. a) Analysis of Provisions of Workmen's Compensation Laws and Discussion of Coverages, prepared by the Insurance Department, Chamber of Commerce of the United States, Washington, D. C., January 1948
b) Personal communication from Mr. Theodore G. Waters, Baltimore, Maryland
3. Kemmer, A. G. Contribution of Industrial Medical Records, Occupational Medicine, 4: 271-273, September 1947
4. Lattes, Leon. Legal Aspects of Industrial Medicine, Industrial Medicine and Surgery, 19: 4-6, January 1950
5. Larsen, Arthur. Workmen's Compensation Law, Matthew Bender & Co., New York City, Vol. II, p. 82, 1952
6. Detweiler, George H. Borderline Compensation Cases, Best's Fire and Casualty News, pp. 61-64, February 1941
7. Watkins, W. Warner. The "Last Straw" Factor in Low Back Disability. Radiology, 52: 20-23, January 1947
8. McGee, Lemuel C. Compensation for Occupational Disease: The Industrial Practitioner's Point of View, Gardner Memorial Volume - Pneumoconiosis, Paul B. Heber, New York City, 1950, Chapter 34, pp. 510-521 (presented at the Sixth Saranac Symposium, 1947)
9. Waters, Theodore G. Need in Workmen's Compensation for Scientific Proof in Medico-legal Controversies, Occupational Medicine, 4: 274-279, September 1947
10. Kyslop, George H. Effects of Electrical Injuries with Particular Reference to the Nervous System, Occupational Medicine, 1: 199-236, March 1946

Discussion

MISS DONLON: In regard to the functions and value of medical boards and of medical examiners it seems to me there are ^{in general,} three different systems that have been organized. One is the plan found faulty in its constitutionality in Michigan and in Pennsylvania, ^{as} and ^{as} long as we have due process clauses I suppose we ^{prefer to enact} ~~shall wish to change~~ our legislation in such a way that it will not run the risk of infringing on the due process guaranteed to our citizens. In the second system there is, ^{Dr.} as Doctor Mayer explained, the kind of board we have in New York, which is a permanent board in that it gives part time to these problems all the year and therefore ^{has a considerable} ~~gets~~ a background of experience from them. ~~And~~ Thirdly, there is the Ohio board, ^{as} presented by Mr. Gentholtz, which is drawn from a large panel - I believe he said 500 physicians. We should remember that in Ohio all insurance under workmen's compensation, except self-insurance, is under a state monopoly system and therefore there ^{is not} ~~isn't~~ quite the ^{same} risk of finding people who are not wholly impartial because ^{of retainers} ~~they have been retained~~ by the insurance companies.

MR. WATERS: My primary practice is in Maryland but I would like to say a word about my practice before the silicosis board of West Virginia. Under the procedure of that board claimants are sent to Charleston where they are hospitalized for three days under the direct supervision of the members of the silicosis board. Physicians on behalf of the employer and of the claimant are permitted to participate in the examination and to testify before the board with respect to his condition. The silicosis board then makes its report to the Commissioner and the Commissioner, after a formal

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hearing in which the claimant and his doctors and the employer and the employer's doctors are heard, renders a decision. My practice there hasn't been much more successful than it has been before my good friend, Dr. Nathan Herman, who is present at this symposium, but the point I wish to make is that the members of the silicosis board do participate as examining doctors of the claimant and the universal practice before the board is for the Commissioner to sustain the findings of that board. I happen to be one of those attorneys who have, on occasion, taken their opinions to the courts, to ^{my} their sorrow.

Chapter Thirty^{two}~~-one~~

The Presentation of Medical Evidence in Controverted Compensation Cases

Remarks by Daniel C. Braun, M.D.

During my tenure as medical director.....
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.....after all, he was under oath!

**THE PRESENTATION OF MEDICAL
EVIDENCE IN
CONTROVERTED COMPENSATION CASES**

by

Daniel C. Braun, M. D. *

Medical Director, Industrial Hygiene Foundation, Mellon Institute,
Pittsburgh, Pennsylvania

During my tenure as medical director of a large corporation, I several times had the experience of watching my medical colleagues spend some very uneasy moments as witnesses in compensation hearings. I knew that they were of the highest caliber, capable, and esteemed in the profession. Yet, placed in the position of expert witnesses in a controversial situation, they allowed themselves to do and say things which were often embarrassing to themselves and to others. Most of this embarrassment arose, I would say, through failure on the part of the physician to observe certain fundamentals.

A basic consideration in the presentation of medical evidence is that the real purpose of the hearing is to bring about a just and equitable adjudication of the claim. If the medical witness never loses sight of this concept, he will not permit his position to become one of zealous partisanship. Such is not the proper function of the expert medical witness. He has a clear duty to testify

*To be presented at the Seventh Saranac Symposium, Saranac Lake, New York September 26, 1952.

when called and to present the medical facts as clearly and concisely as possible in order to aid the court in the proper interpretation of these facts. It should be obvious that this function is of the utmost importance in establishing whether the claimant has been disabled as the result of an accident or occupational disease, and the nature and extent of such disability. The performance of this duty requires from the medical witness the same high level of integrity as does the everyday practice of his profession.

If we presuppose adequate training and experience on the part of the physician, there remain two main requisites for proper medical testimony. The first is adequate preparation of the case. However well trained in the profession and in his speciality, the witness is not necessarily an expert in the case at hand. I have never quite understood why so many doctors assume that they can dash into court and testify extemporaneously. It seems to me that for his own sake, and in justice to the claimant, he should be willing to prepare his testimony with the same careful attention to detail with which the lawyer prepares his case. Admittedly this horrible example which we are using generally has with him the hospital records which he nervously leafs through while mumbling his testimony into his lap, which in itself detracts from anything he may have to say. Really adequate preparation of medical testimony requires that the physician keep complete records on the case from the beginning. All cases of personal injury or illness arising in the course of occupation are of the type which have what Kammer¹ has called a "high medico-legal potential". In such cases he lists the following minimum components of a

full medical record: (1) a record of the physical status of the person before a specific event (accident or exposure), (2) an accurate description of the event, and (3) a record of subsequent changes in the individual. I am sure that by this he means "including adequate and frequent x-rays", but the importance of x-rays at the time of accident and during the progress of the case needs to be stressed. Certainly with such records the medical witness should not be in the embarrassing position of relying on his memory or contradicting his own statements with respect to the record.

Helpful as the medical records may be, they constitute only a portion of the preparation for testimony. Hypothetical questions are frequently put to the expert witness and he should give adequate thought beforehand to the possibilities of such questions and the answers, based on a reasonable chain of causation.

In this connection, it would be well for the physician who may be called as a witness to consider the case from a broad viewpoint. Instead of confining his thinking to the type, location, fixation, and union of a fracture, he should regard the person who sustains the fracture as a complete individual--the effect of the fracture on his future physical and emotional status, and the possible connection with preexisting or coexisting pathological conditions. One of the obstacles to good testimony, and therefore to a just disposition of the case, is the frequency with which witnesses are caught off guard by a question which they did not anticipate because their thinking was too narrowly circumscribed. For example, consider a situation in which a man dies while working in an atmosphere known to contain some percentage of carbon monoxide.

An autopsy shows beyond question that death was due to coronary occlusion. The medical expert testifies that coronary artery disease was the proximate cause of death and that death was inevitable because of the pathological condition. Various questions are asked him concerning the conditions which exist in the coronary arteries and which lead to a coronary occlusion. He discusses the changes in the arterial lumen and resulting anoxia. Suddenly he is asked whether a man with this set of conditions would not be more likely to have a heart attack if a portion of his hemoglobin was saturated with carbon monoxide and hence not available for transportation of oxygen to the tissues. Possibly because he has not acquainted himself with the effect of various concentrations of carbon monoxide in the blood, or because he failed to determine the level of carbon monoxide in the blood in this particular case, or because he failed to inquire into the concentration of carbon monoxide in the environment, he finds himself in a weakened position and is gradually forced to admit that carbon monoxide may have aggravated the man's condition. This exact situation may seem to be exaggerated, but it is not. It illustrates the necessity of acquainting oneself with all the information available. It may be necessary that, in the preparation of a given case, the physician review the latest literature in order to apprise himself of the newest information.

A second requisite of good medical testimony involves the conduct of the witness, and this, too, will be improved by virtue of proper preparation much as outlined above. He must not only be scrupulously honest in his testimony, but his deportment must be such as to impress the referee or the jury that he is

honest. He should, of course, conduct himself in a gentlemanly manner on the stand and refrain from flippancy and argumentation. To allow himself to become bombastic or pompous quickly indicates to the court that he is not sure of his ground and just as quickly leads to retaliation in kind by the opposing attorney. Any witness will improve the quality of the medical testimony and add to his own composure if he will remember to take time to be sure that he understands the question directed to him and the implications of his answer before formulating his reply.

In the matter of actual testimony, it is important to face the referee or jury and to enunciate clearly and distinctly. One of the most common criticisms (and probably the most justified) of medical witnesses is their tendency to testify in the jargon of the medical textbooks. "The language of the craftsman must be made intelligible to those untutored in the craft. The compensation referee or commissioner in evaluating the medical testimony should be aided by the use of simple medical language whenever the occasion offers."²

While it is certainly unwise to make dogmatic statements especially in hypothetical matters, such as those dealing with aggravation of preexisting conditions, one of the things most deplored by lawyers is the lack of a definite expression on the part of the medical witness when such might be expected of him. If he is, in fact, an expert he should be prepared to state his opinion and not resort to vague terms such as "probable" or "possible". The same vagueness also frequently appears in support of a witness's estimate of percentage of disability. I believe that the disability should be based upon some reasonable standard, although I will admit that opinions vary widely in this respect.

In most cases of this kind, the x-ray films play a very important part. The witness should familiarize himself with the rules regarding their admission as evidence in the particular jurisdiction. Films must be properly identified. Their interpretation should be regarded as a privileged communication. The medical witness should remember the limitations of the x-ray and should be frank to say so if the findings are equivocal. He should properly correlate the x-ray and clinical findings without attempting to over-emphasize the x-ray, or, on the other hand, to make it conform to the clinical aspects of the case. In the matter of chest x-rays, it is important to remember that, to be of value, they should be made under certain specified conditions of technique. The length of exposure should not exceed $1/10$ of a second, for example, and the film-target distance should be not less than 4 feet.

I do not wish to leave the impression that most doctors are poor witnesses or that all the difficulties arise because of their shortcomings. The evidence in such cases is bound to be controversial and it is expecting a great deal to ask that the physician remain entirely objective. There are other reforms which could be made and which would contribute greatly to the obtaining of good medical testimony in controverted compensation cases. For one thing, there is a need for more training in medico-legal disciplines in our medical schools. Legislation should give greater consideration to the value of neutral medical examiners or medical boards, such as the Long Shoremen's Act provides. Waters³ has consistently pointed out the value of such impartial testimony, and 22 states now provide for medical boards. The pernicious practice of one side or the other employing a physician who is willing to testify

for the highest bidder can be minimized by the medical society and the bar association concerned working together in the manner of the Minnesota plan on medical testimony⁴. This joint effort of the Minnesota Judicial Council and the State Medical Association results in a review of the testimony given by a medical witness, if such a review is requested by the judge, or a physician or an attorney representing either side. The review is done by a committee of physicians who are aided by three specialists in the particular field involved. It has resulted in more careful and honest testimony on the part of medical witnesses. Another great need is for standard criteria on which to base disability evaluation.

In closing, I believe that the most important single requisite for the presentation of medical evidence is the scrupulous honesty of the witness. This, I think, can be illustrated by an amusing story which is told of Dr. Henry Rowland, then Professor of Physics at The Johns Hopkins University. During his suit against the public utilities companies, he was asked by the celebrated Joseph H. Choate, in cross examination, "Who is the greatest living physicist?" Dr. Rowland unhesitatingly replied, "I am". Afterward, when asked by President Gilman of Hopkins why he gave that answer, he smilingly replied that it was a little embarrassing, but that, after all, he was under oath!

LITERATURE CITED

1. Kammer, A. G., M. D., "Contribution of Industrial Medical Records," Occupational Medicine 4:271, Sept. 1947.
2. Edson, George N., M. D., "Obtaining Qualified Medical Testimony," Occupational Medicine 4:267, Sept. 1947.
3. Waters, Theodore C., LL. B., "Need in Workmen's Compensation for Scientific Proof in Medico-legal Controversies," Occupational Medicine 4:274, Sept. 1947.

Waters, "The Lawyer and the Doctor," Compensation Medicine, June 1949.
4. Workman, W. G., M. D., "A Committee on Medical Testimony," Occupational Medicine 4:280, Sept. 1947.

Remarks by Frank R. Parnako

You will note.....

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.....be assisted and expedited.

Revised

THE PRESENTATION OF MEDICAL EVIDENCE IN
CONTROVERTED COMPENSATION CASES

By

F. R. BARNARD
Manager, Compensation & Safety
Bethlehem Steel Co.

You will note, from the introduction, that I am an employee of a large industrial concern. Hence, quite naturally, my viewpoint in discussing controverted compensation cases is that of the defendant. I think you will agree that it could hardly be otherwise, although I am not without experience on the plaintiff side; having spent some years in general practice prior to employment with Bethlehem Steel Company. In order that my remarks may be of general application and interest, I have tried to analyze the problem presented here from both the plaintiff and defendant standpoint. In addition, since this symposium is concerned with health problems pertaining to workers exposed by inhalation to industrial substances, my discussion is principally concerned with the presentation of evidence in that type of case.

I take it that "controverted compensation case" applies to a claim for disability alleged to be the result of employment; payment of which under the compensation law has been denied upon the considered judgment of the employer or carrier involved, but only after full investigation of all facts, including medical consultation and opinion.

Although this audience is mainly composed of attorneys, physicians, and others specializing in compensation work, it would seem advisable to establish the meaning of a few of the terms contained in this topic as

selected for this panel. Those which I have in mind are "presentation", "evidence", and "medical evidence". These are defined as follows:

- (1) Presentation - "Act of presenting or state of being presented".
- (2) Evidence - "That which tends to prove or disprove any matter in question or to influence the belief respecting it. Belief is produced by the consideration of something presented to the mind. The matter thus presented in whatever shape it may come and through whatever material organ it is derived is evidence. Testimony is not synonymous with evidence; evidence is a more comprehensive term and includes statements of witnesses, contents of papers, documents or records. Evidence may be considered with reference to its instruments, its nature, its legal character, its object, or the mode of its introduction".
- (3) Medical Evidence - "Testimony given by physicians or surgeons in their professional capacity as experts or derived from the statements or writings of medical or surgical works".

Parenthetically, the word, "opinion" may be defined in a general way as "a belief, a view, judgment which the mind forms".

You will note that although presentation of evidence, by definition, refers to the procedure of placing evidence before a hearing body, the purpose of evidence is both to "prove a matter in question" and "to influence the belief respecting it". Certainly, we can agree that in many compensation cases, and most occupational disease cases, medical questions are not only of primary importance but, frequently, are the main subject in dispute. For the answers to such questions, we must rely on the opinions of physicians and from such opinions and other evidence the hearing official then forms his own opinions and makes his findings.

The reason evidence is presented in compensation or other litigation is, therefore, to provide a basis for the trial agency to form a belief or opinion on the affirmative or negative side of the facts in dispute. To convince the commission or court that the side which he represents is the correct one is, then, the ultimate objective of the lawyer in presenting the evidence. It is axiomatic that a disputed subject or question is best presented by a person thoroughly familiar with all phases of the issue and convinced of the validity of his arguments. In addition, the evidence must be supported by facts stated in a logical and concise manner. It is fundamental that to convince another you, yourself, must be convinced of the truth of your position.

The presentation, argument or, as in the subject under discussion, the medical opinion, is but the conclusion in a chain of events which begins with study, investigation, and compiling of all material facts. It is my belief that litigation is most frequently won or lost at this stage. We have all heard or read of brilliant cross-examination of a hostile witness or a strategic admission or objection which changed the result of litigation. There is no doubt that these events have occurred, but that they are most generally the product of long-range planning, painstaking study, and thorough familiarity with the case at issue is equally true.

Originally, compensation laws were enacted as a method by which informally and without the delay and difficulties of court procedures, the question of payment for industrial injuries and diseases could be disposed of. Since that time, however, judicial interpretations, in-

creased medical knowledge, the advance of research and science resulting in the evolution and use of new processes and materials in industry, have all contributed toward making many of the cases presented today most difficult to decide. Diseases which were unknown when compensation laws were originally enacted are being compensated today as, for instance, the disease which is the result of exposure to barrylin. Indeed, in those states where they exist today, occupational disease laws are generally of comparatively recent enactment. Furthermore, in states which have enacted an "all-inclusive" statute, the issue of whether or not the disease claimed for is an occupational disease involves mixed medical and legal questions, frequently requiring appellate court determination. Anyone could, of course, amplify these examples of the problem presented. Suffice to say that no controverted compensation case can be "tried by ear" and there is no question that, for reasons obvious to all of us, such a thing should never occur.

Yet, who is not familiar with the attorney who, upon receipt of a written report from the physician to whom he has referred his client, prepares for the hearing by calling the doctor and asking, "Doctor, I have your report. Is this what you're going to testify to on the stand?" Despite the fact that this is a foolish question, the doctor without further comment replies that he will so testify. The attorney then says, "Thank you" and "Goodbye" with the statement, "Fine, Doctor, I'll see you at the hearing." Far too often this is the full extent of conversation between the physician and attorney until the physician takes the witness stand. Yet, that same attorney will criticize the physician after the hearing for

not having given the proper answers to his poorly prepared questions.

Conversely, we have all experienced the doctor who appears at the hearing with only half, or even none, of his notes of examination or supporting data for his opinion; which fact may not be discovered until he has already begun to testify. Neither of the foregoing events should ever occur and their prevention is the joint responsibility of attorney and physician.

In those cases wherein medical opinion is a deciding factor, the responsibility for the determination of the case and whether or not the claimant will procure an award, rests upon the attorney and physician. This is particularly true in occupational disease cases. In the solution of the questions presented, the attorney and physician cannot act as individuals, each responsible for his own specialized field of training. The attorney and physician are a team. Too frequently, attorney and physician seem to feel that they must zealously guard the prerogatives of their professions and that the exchange of advice and experience is a sign of weakness and ignorance. It cannot be gain said that the complete and thorough cooperation between the attorney and physician is most essential to the end that:

- (1) Both thoroughly understand the medical and legal issues involved.
- (2) Both know the evidence available which is to be offered; the manner of its presentation; together with supporting authorities.
- (3) Both are agreed upon the purpose of the evidence and what allegations it supports.

- (h) The subject of direct and cross-examination is thoroughly discussed so that preparation can be made for the proper questioning of medical and even lay witnesses.

A physician who does not know the type and quantum of proof necessary to fulfill such statutory requirements as exposure and causation cannot be expected to meet those issues squarely with his testimony. Even legal questions such as the statute of limitations may depend solely upon the history which has been taken by the physician in the course of his examination of the claimant. By the same token, an attorney cannot develop the testimony and present it unless there has been made available to him complete reports, including occupational history, symptoms, physical findings, diagnosis, and disability. It is also vitally important that the physician should accumulate and discuss with the attorney those records which sustain or corroborate his medical opinion.

Not infrequently, litigation will involve one or two vital questions which may be decisive. In occupational disease cases, where the primary issues are usually those of causal relation, type and nature of hazard, and extent and nature of disability, those questions depend on the medical evidence. When, through conferences between attorney and physician, these problems are resolved and their relative importance is agreed upon, the attorney is in position to decide which are vital and, thus, is able to chart his course in the case so that effort and time will not be expended on irrelevant matters. Emphasis on the essential questions will also serve to direct the attention of the hearing commissioner toward the most important issue.

In the majority of cases, the examination of the claimant by the physician is a vital part of the testimony since it is the basis upon which the physician expresses his opinion. Much could be said on this subject, but it is not properly a part of this paper. However, I do wish to point out that it is not sufficient that the plaintiff has been examined by the physician and as a result of his findings he is prepared to testify to the existence or non-existence of a claimed disease or other disabling condition. The attorney and physician should be certain that, insofar as possible, this examination not only sustains the position taken with respect to the cause and existence of the claimed disability, but that an alternative diagnosis has been made. Failure of a physician to examine an alleged silicotic for heart involvement or other vascular conditions can be embarrassing on cross-examination and sometimes decisive.

The examination in chief of a physician should receive special attention. The attorney and physician should confer on the questions to be directed to the physician so that he will understand what he is expected to testify to and what is required of him. In turn, the physician will be able to assist the attorney relative to the questions which should be asked and the manner of their phrasing so as to best develop the medical testimony. Similarly, weaknesses in the opposing medical opinions should be discussed so that the attorney will be able to conduct advantageous cross-examination. Study of the literature on the disease alleged in support of, or contrary to medical opinion to be presented by both parties, will facilitate direct examination and will furnish material for cross-examination.

Since the definition of medical evidence given previously includes "statements or writings of medical or surgical works", authoritative writings are also of assistance in supporting medical opinion to be offered and may, of themselves, be used as evidence.

As a result of this preparation and investigation, the medical evidence is now ready for introduction at the hearing. Its purpose is to provide the administrator with information from which he can form an opinion. The attorney, however, must keep in mind that while he is familiar with the facts of the case, the hearing commissioner is not. Therefore, the evidence must be presented in such a manner as to assure that he will understand and be convinced by it. Evidence which is disjointed, interrupted and uncoordinated is difficult to comprehend, hence, efforts should be made to present the evidence in a logical and even chronological sequence. For instance, presentation of evidence on the issues of cause and extent of disability before evidence of exposure or non-exposure to the alleged hazard, is not a logical sequence and tends to confuse. So that clarity may be effected, some attorneys have found it beneficial to prepare a check list of the evidence and issues in order to assure that testimony is presented on all matters in dispute and in proper sequence.

The nature and extent of the hazard to which claimant alleges he was exposed is an essential element in occupational disease cases. Proof of exposure or non-exposure does not usually involve the testimony of physicians since such evidence is supplied by industrial hygienists, chemists, etc.; but it does not follow that the medical evidence is in

no way concerned with the details of the claimant's occupation.

As a result of previous personal experience or information otherwise acquired, people frequently assume the existence of a hazard or exposure merely from the location of claimant's work or description of his job. This is true of both attorneys and physicians and sometimes of compensation administrators. The fact that the claimant is a "chipper" in a steel plant does not mean that he is exposed to an occupational hazard or that his disability is the result of employment by the defendant. Many "chippers" in the steel industry never saw the inside of a foundry nor are they employed in any operation involving silica-bearing materials. It is, therefore, suggested that the attorney and physician familiarize themselves with the claimant's work and make certain that in interviewing the claimant he has given a full description of his employment activities. It is quite possible that the extent and nature of the hazards of the occupational process will be described in varying forms by claimant and defendant witnesses. Preparation along the lines above suggested allows the parties to explain and clarify misleading descriptions and tends to remove much of the mystery and preconceived notions held by many individuals unfamiliar with industrial occupations. A concomitant of such testimony is that the medical evidence becomes more understandable and the hearing commissioner is assisted, since he is able to form a mental picture of the basic element of the claim which, after all, is the occupational process the claimant and the industry which employs him are performing. At the same time, such preparation and testimony furnish excellent opportunities for effect-

ive cross-examination.

Dr. Braun has ably discussed the subject of physicians' testimony as differentiated from the introduction of medical evidence. However, I would like to direct a few remarks to attorneys on this subject. I have already stated that testimony is best understood when presented without interruption and in logical sequence. In connection with a physician's direct testimony, questions which require interpolations, repetition of prior statements, or explanations tend to detract from the value of the testimony. Therefore, his evidence, including records, x-rays, etc., should be presented with as little interruption on direct examination as is possible. If attorney and physician have properly prepared themselves, this can be accomplished. Needless to say, the time required for the hearing can also be diminished in this way.

There is a common misconception of the purpose of cross-examination. In my opinion, it should never be undertaken without a specific purpose in mind. Experience indicates that seldom, if ever, is a physician's opinion changed through cross-examination; but, on the contrary, if the opposing physician is properly prepared and is experienced in testifying, lengthy cross-examination without purpose will merely present him with the opportunity to strengthen the opinion to which he has already testified. Certainly, a cardinal rule of cross-examination is "DON'T".

You will, of course, appreciate that what I have said in this paper is personal opinion and, furthermore, is not exhaustive of the subject. Its purpose, rather, has been to highlight some of the essential

elements and to provide a basis for discussion. Certainly, the subject is of importance and one to which all those concerned in the operation of the compensation law must devote time and study. To the extent that compensation cases are competently tried, to that same degree will the work of the commissions in disposing of controverted cases be assisted and expedited.

Remarks by J. H. ^{Harry} Tiernan, Jr.

It is my privilege today.....

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.....in silicosis cases.

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~~MADAM CHAIRMAN, LADIES AND GENTLEMEN:~~

It is my privilege today, to outline the procedure followed by the United Steelworkers of America, Dist. 4, and its counsel in presenting a claim for disability due to silicosis.

A knowledge of the purpose for which a medical report is made should be of material aid in its preparation.

The claim is made pursuant to Sec. 3 Sub. 2, Par. 28 of the Workmen's Comp Law, effective July 1, 1947. Certain specific requirements must be met which differ materially from the ordinary.

The claim is properly initiated by the preparation and filing of A C 3 form entitled "Employees Claim for Compensation" and such filing causes the Board to refer the claimant to the Board of Chest Consultants for examination and opinion. Upon receipt of the Consultant's reports the case is scheduled for hearing.

Claimant's attorney must then proceed to prove the claim. Such proof in a silicosis claim requires that two major factors be established that need not be asserted in the ordinary Workmens case.

First: Exposure to injurious dust within two years prior to disability.

Second: That claimant is totally disabled.

As to the first factor, the claimant is aided by the provisions of Sec. 47 of the law which reads as follows: "Any exposure to the hazards of harmful dust in this state for a period of sixty days after Sept. 1, 1935, shall be presumed, in the absence of substantial evidence to the contrary, to be an injurious exposure."

Thus it is possible, merely by having the claimant testify to the nature of the dust, to establish whether or not it is injurious, and it is then the obligation of the carrier to offer substantial evidence that such dust, for a variety of reasons, may not have been injurious. Commonly the carrier offers evidence that the dust may not have contained silica or other injurious matter, that the concentration was of too low a dust count, or the particles too large.

It is most fortunate that Section 47 is in the law, for otherwise it would be almost impossible for the claimant to prevail. No dust counts or particular size measurements are ever available to the claimant who never has occasion to consider the matter until after the effect has been felt. On the other hand, the company may at any time have its plant inspected and have reports available.

Usually, however, such counts are not made until after the claim is filed and unless the carrier or employer can establish that the conditions under which the claimant worked were substantially the same as at the time of inspection, the presumptions as to injurious exposure will not have been overcome and claimant may then proceed to the question of total disability.

This is primarily a medical question. Present medical opinion appears to be that silicosis per se, is not disabling. Any disability is evaluated on the basis of complicating factors. The attorney, therefore, must be able to establish by competent medical proof either that the condition is complicated by infection, emphysema or cardiac involvement.

It is most important, therefore, that the attending physician, in reporting to the board, investigate and detail such complicating conditions and be prepared to support his opinion. It is not necessary that the dust disease be the sole cause of the disability but it must be at least a contributing factor. Furthermore, unless the physician is able to conclude that claimant is totally disabled the proof must fail.

In evaluating the degree of disability it is usually necessary to take into consideration factors other than the immediate effects of the disease itself and its complications. The age of the claimant is an important factor as are other diseases from which he may suffer.

It is proper for the physician to state that even though the silicosis in and of itself plays only a part in the entire picture, that nevertheless, if it is a contributing factor to the total disability, then the condition is at least in part related to the occupation.

I have mentioned infection, emphysema and cardiac involvement as complicating factors in silicosis. These appear to be the only conditions presently considered as complications. We who have represented the United Steelworkers of America in the Buffalo area have observed a significant number of cases in which individuals exposed to silica have also been found to be suffering from carcinoma of the lung.

No physician has yet expressed the opinion that exposure to silica has any connection with this condition. We hope that future studies may throw further light on the question.

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We also believe that compensation should be awarded for partial disability. We do not claim that the exact degree can ever be determined, but the exact degree of partial disability never can be evaluated in any case. It should be enough that silicosis, even though not totally disabling, effectively destroys the earning capacity of a stonecutter, molder, bricklayer or other mechanic in his skilled occupation and forces him to seek employment at lower wages in other occupations. We hope therefore, that there may soon be a realist re-evaluation of this problem.

On behalf of the United Steelworkers of America Dist. 4, I thank Dr. Vorwald and Miss Donlon for the opportunity of explaining a few of the problems of our members and of outlining briefly our objectives in silicosis cases.

J. HARRY TIERNAN, JR.

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Remarks by Ivan Sabourin

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MR. KNIGHT: From all that I have heard here and from my experience of 42 years with the Compensation Act and as chairman of the Illinois Industrial Commission I am heartily in favor of an advisory medical board, the board in our state of Illinois to be chosen by the medical colleges within the state, among them being the University of Illinois, ~~Bethel College~~, Northwestern and Chicago. I believe that we who have no mandatory occupational disease act are practically helpless at times in the proper administration of that act. If the medical men can't be of assistance to us, I know of no one who can.

DR. HERMAN: It would have been interesting and possibly useful to have heard something about the disadvantages of medically-trained boards or technically-trained boards in arriving at substantial justice. Being a physician and a member of a board that is constituted somewhat differently from those of other states, I am, of course, heartily in favor of the medically-trained board.

I believe that Dr. Braun's remarks are particularly pertinent since frequently physicians appearing before us are ill-prepared in the testimony they give and their records are inadequate. If the requisites of good medical testimony were more generally recognized by physicians, medical administration would, as Dr. Braun pointed out, be much simplified.

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What does this prove? It proves how difficult is the situation of workmen's compensation administration in the face of medical opinion so diametrically opposite. The handicap is that of a law which places great restrictions on the right of the claimant. Had we a law in this state which permitted a finding of disability for a partial disability, perhaps we would not find competent doctors straining quite so hard as they do to find total disability.

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WORKMEN'S COMPENSATION COMMISSION
LANSING 13, MICHIGAN

JAMES L. HILL
COMMISSIONER

September 12, 1952.

Arthur J. Vorwald, M.D.,
Director, The Trudeau Foundation
and The Saranac Laboratory,
P. O. Box 551,
Saranac Lake, New York.

Dear Doctor Vorwald:

Enclosed are two copies of my proposed
remarks for the panel discussion. I have also sent
a copy to Mr. Theodore Waters.

I am looking forward to meeting you
and participating in the Symposium.

Sincerely yours,

James L. Hill

JLH:PK

Official copy

As originally enacted none of the workmen's compensation acts specifically provided for the payment of benefits for disability or death resulting from occupational diseases. However, the Massachusetts Act passed in 1911 covered personal injuries without the qualification that they be accidental but it was not until 1929 that it was determined that silicosis was covered by the law. It is now generally recognized that disability or death from occupational diseases should be compensated. Occupational disease coverage is provided in 41 States, Alaska, District of Columbia, Hawaii, Puerto Rico and under the Federal Employees' Compensation Act and the Longshoremen and Harbor Workers' Act. General coverage for all occupational diseases is provided in 24 States, Alaska, District of Columbia, Hawaii and under the Federal Employees' Compensation Act and the Longshoremen and Harbor Workers' Act. Schedule coverage is provided in 10 States and Puerto Rico. It is quite obvious that the all-inclusive type of coverage provides the better protection and the only just coverage. There is not one good reason to give compensation benefits to one man suffering from an occupational disease and denying them to another simply because the latter is suffering from a disease not known when the schedule was made.

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We have a similar situation when a foundry lays off men in a slack period or ceases operations and when a mining company closes its mine. The foundry worker with abnormal chest findings simply cannot get a job

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Should the compensation laws contain a provision that to be compensable disability from silicosis must occur within a specified period after the last exposure or after the last day of work for the employer from whom compensation is claimed? I say, absolutely not, but several States have such a limitation in their compensation law varying in time from 120 days to 2 years. Should a man become disabled from silicosis 25 months after he has, for one reason or another, left the employment in which he was exposed to silica dust, he would not have a remedy in many of our States in which silicosis is supposed to be compensable. His right to compensation would be barred because of the lapse of time since exposure or employment. There is no good reason for denying compensation merely because the worker's disability did not occur within a specified period after exposure when the cause of the disability is established. Here in New York a worker, or in case of death, his dependents, need only file a claim within 90 days after disablement and after knowledge that the disease is or was due to the employment. The only time limitation in

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Workmen's compensation for silicosis has created several problems, none of which can be solved by an assortment of limitations manifestly unfair to the workers. I believe the old adage, "an ounce of prevention is worth a pound of cure", is apropos in this field. Most of the problems can be solved by preventive measures designed to eliminate or minimize the silica hazard and by realistic and sensible employment standards. The duration of the problems will depend directly upon the willingness or unwillingness of employers to adopt such measures and employment standards.

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Remarks by James L. Hill

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Mr. Hill
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ADMINISTRATION OF WORKMEN'S COMPENSATION LAWS AS THEY RELATE
TO THE PNEUMOCONIOSES

Moderator: Theodore C. Waters

Panel Members: James L. Hill
William L. Connolly
B. E. Keuchle
Earle T. Andrews
Lloyd E. Hamlin, M. D.

SUMMARY

Workmen's Compensation Sessions

Theodore C. Waters

CONCLUSION OF THE SYMPOSIUM

Summary of the Medical Sessions and Remarks

Arthur J. Vorwald, M. D.

(2:30 to 5:30 P. M. - Sept. 26, 1952)

BY MR. WATERS:

Gentlemen, will the gentlemen in the back part of the room kindly come forward and be seated. I have two announcements to make. Doctor Vorwald has requested me to announce that Doctor Alvin A. Rush will be driving to Michigan by way of Watertown and Rochester. He has available room to take care of two passengers and will be very glad to take two passengers with him. Doctor Rush, I believe is in the audience and if anyone would be interested in that invitation, please speak to him promptly.

The second announcement is that, due to airplane schedules this afternoon, we must conclude the afternoon session by 4:30. I'm very sorry to have to tell you that, but several or two of the participants in the panel have reservations on that plane and I understand that several of the members in the audience are in a similar situation.

Well, we come now to what I believe is and should be one of the most interesting sessions of the Symposium, dealing with the panel upon the subject of the Administration of Workmen's Compensation Laws. This panel deals with the laws primarily as they relate to the compensation for the pneumoconioses.

The parties in interest subject to that administration, are four, the employee, the employer, the insurance carrier and the public. Industrial workers have their primary contact with the administration of American jurisprudence before our various compensation commissions, and the successful administration of these laws is a matter of concern to all of the four parties whom I have just identified.

^{1-1/2} One of the speakers this morning referred to the fact that more claims came before our compensation commissions than any combination of claims or suits that come before our law courts, so I think that that statement is very significant, and may I say just a word with respect to our procedure this afternoon.

I shall introduce each participant in the panel and he will make a brief statement as to his point of view in the administration of the law. This will be followed by the presentation of prepared questions to the participants in the panel. We feel that that will better enable them to develop their subject and their discussion to your interest. Following the prepared questions, the audience will be invited to direct questions to the panel and to participate in the discussion. No one has been told what to say and what not to say. This is an open forum. It is your meeting as members in attendance at this Symposium.

Even though we may disagree, and I happen to know that among these five gentlemen sitting up here, there is substantial disagreement with respect to various features of the administrations of the law, we still expect the individual opinions so expressed, and we hope that the discussions presented will lead to an open and frank discussion of the various phases of the subject.

May I ask those in the audience who desire to present questions, to do so in a form as simple and direct as is possible. Bear in mind that this subject today is subject particularly to limitations of time. It is hoped that when these questions are directed to the panel, not one but several of the members will participate and join in their answers, but limitations as to time require that not more than two minutes be given to those answers, and your

cooperation in that respect will be deeply appreciated.

The first speaker upon the program has served with distinction as a member of the Workmen's Compensation Commission of the State of Michigan for many years, and in that capacity, he has served a distinct public office in the administration of his job. Compensation laws are complex. We lawyers happen to make them so, both as they relate to the letter of the law and its interpretation, and they have presented peculiar problems to those who are charged with their administration.

The State of Michigan, with its vast industry, has experienced all of those problems that we have been discussing here during the course of this symposium. James L. Hill became Legal Advisor to the Commission in 1936. Therefore, you recognize the length and the importance of his service. Since that time, he has been serving as one of its members. He is also a past president of the International Association of Industrial Accident Boards and Commissions and brings to us a wealth of experience, not only in the formulation of legislation, but in the administration of the laws. It is my pleasure to introduce to you, Commissioner Hill.

BY COMMISSIONER HILL:

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Mr. Chairman, Ladies and Gentlemen: It's an honor and a privilege to be here. I'm grateful for the opportunity

to participate in this symposium. I'm tremendously impressed with the amazing job that is being done here and I only wish that all compensation administrators could have this experience, as I am sure that it would be as inspiring to them as it has been to me.

As originally enacted, none of the workmen's compensation laws specifically provided benefits for disability or death resulting from occupational disease. However, the Massachusetts Act, passed in 1911, covered personal injuries without the qualification that they be accidental, but it was not until 1929 that it was determined that silicosis was covered by the law.

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Now, I was not here on Monday morning, so I didn't have the benefit of the definitions, but as I understand the term 'pneumoconiosis' it means dust storage and includes all conditions of the lungs that result from the inhalation of dust, that from simple dust storage and those from the storage of various irritating dusts, the most common of which is silica.

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Some physicians say the emphysema is secondary to

the aging process and not to silica storage when the X-ray film fails to disclose nodular fibrosis. They maintain there must be nodular fibrosis in order to have sufficient lung involvement to cause emphysema. Other physicians take a contrary view and state that where the X-rays show increased linear markings, together with emphysema, that the emphysema is caused by the lung involvement due to silica storage.

Is it possible to determine which medical theory is accurate? The answers to these questions, if they can be specifically answered, would be invaluable to the compensation administrators who are not fortunate enough to have an impartial medical board to make the medical determination. It seems quite obvious that the most satisfactory result can be obtained by having the medical determination of whether the claimant has silicosis or any clinical dust disease conclusively decided by a qualified impartial medical board.

A large percentage of our silicosis claims in Michigan arise because the unemployed foundry worker or miner is unable to get a job. It is the policy in some foundries to discharge a worker when his chest X-ray shows an abnormal condition. That policy is not used by a single mining company. The worker is invariably refused employment when he applies for work at another foundry.

Generally, he has had several years experience in a skilled occupation and knows no other trade. He is willing and able to continue working at his trade, but his training, experience and ability to work are of no value to him when he can not get a job. Having failed to get a job, he employs an attorney and a silicosis claim is filed. It's not unusual for the physician who recommended his discharge to testify that the man does not have silicosis, because the disease can not be recognized from chest X-rays.

The claimant's physician will probably testify that the X-rays show first degree silicosis or both may testify that the X-rays show increased linear markings, but one says no silicosis and the other calls it early silicosis.

It makes no difference to the man whether it's discreet nodular fibrosis or increased linear markings. He doesn't care what the physician calls his condition. The important fact, to him, is that he can not get a job at his trade. He may not be physically disabled, but he has a definite disability, because his potential earning capacity has no value to him when he is not employable. His loss of employability must be considered in determining his right to compensation. Physical inability to do the job can not be the sole test.

We have a similar situation when a foundry lays off men in a slack period or ceases operations, or a mining

company closes one of its mines. The foundry worker with abnormal chest findings simply can not get a job, and sometimes the foundry does not recall him for work though the mining companies, invariably provide surface jobs for the men who have silicosis and should not work underground. They will not hire another company's workers when they have abnormal chest findings. These men become compensation claimants because they are not able to secure employment at their respective trades.

What can be done to prevent this loss of employability of workers with abnormal chest findings? How can the needed productivity of these men be utilized? It is certainly unrealistic to put these skilled workers on the shelf and pay them compensation which is unproductive and, at best, a very poor substitute for a regular pay check. Most of them would prefer to continue at their trade.

We are in a vicious cycle, because of the employers' employment standards. Of course, if you want to blame someone, you can readily blame the compensation administrator. We're public servants and it's rather popular to make us a whipping post. However, I think that any fair analysis of the problem will indicate that the loss of employability which precipitates a large percentage of the silicosis claim is due either to the employers' lack of understanding or his refusal to attempt to solve a problem which is peculiar.

within his power to solve.

The industrial physician is not entirely blameless because the employer often acts on his advice. The burdens are not going to be relieved until employers use every scientific measure known to remove the dust hazards and provide safe working conditions as many have already done, and then make any and every possible effort to utilize the skills of every available worker even though he may have some chest abnormality when such condition does not interfere with his performance of work.

Only then will we have a paucity of silicosis claims and the cost bought in human misery and compensation for silicosis will level off to a very minimum.

Several of the compensation laws provide that no compensation is payable for partial disability from silicosis. It would be almost impossible to evaluate percentage-wise a man's loss of physical ability due to silicosis. A silicotic is either employable in his regular trade or in another occupation where there is no exposure to silica or not employable. He should be compensated when he can not work at his regular trade or is unable to secure work because of his silicosis. He should be compensated for his wage loss when he is employed in a non-injurious occupation at a lower wage. He should be paid compensation in the amount of the difference between his weekly wage in his skilled

trade and that in the non-injurious occupation, but not to exceed the maximum payable for total disability.

Should the compensation laws contain a provision that to be compensable, disability from silicosis must occur within a specified period after the last exposure or after the last day of work for the employer from whom compensation is claimed? I say absolutely not, but several states have such a limitation in their compensation law, varying in time from 120 days to two years. Should a man become disabled from silicosis 25 months after he has, for one reason or another, left the employment in which he was last exposed to silica dust? If so, he would not have a remedy in many of our states in which silicosis is supposed to be compensable. His right to compensation would be barred because of the lapse of time since exposure or employment.

There is no good reason for denying compensation merely because the worker's disability did not occur within a specified period of time after exposure when the cause of the disability is established. Here in New York, the worker or in case of death, his dependents, can go and file a claim within ninety days after disablement and after knowledge that the disease is or was due to employment. The only time limitation in Michigan is that notice of the disablement must be given to the employer within 120 days after the date of disablement, which has been construed by our

court to be within 120 days after the worker has knowledge or reasonable ground for knowledge of his disablement.

In at least five other states, any limitation against the claim does not start to run until the worker becomes disabled from silicosis. There is absolutely no reason for making it more difficult to get benefits for silicosis than for a traumatic injury, when the disease is due to the nature of the employment.

Workmen's compensation benefits for silicosis has created several problems, none of which can be solved by an assortment of limitations which are manifestly unfair to the worker. I believe that the old adage, an ounce of prevention is worth a pound of cure, is a proper - is appropriate in this field. Most of the problems can be solved by preventive measures designed to eliminate or minimize the silica hazard and by realistic sensible employment standards. The duration of the problems will depend directly upon the willingness or unwillingness of employers to adopt such measures and employment standards. Thank you. (Applause)

BY MR. WATERS:

Thank you very much, Jim. That was well stated, very effective statement of your point of view. Many of the issues, I happen to disagree with you upon, but that's my privilege and the similar privilege of those in the audience.

Our next speaker, William L. Connolly, is a Director of the Bureau of Labor Standards of the United States Department of Labor. He was called to that post after having served for six years as Director of Labor of the State of Rhode Island where he supervised the administration of the workmen's compensation laws of that state.

To his honor and credit, he is a former president of the Rhode Island State Federation of Labor. Some years past, he has served with distinction, and I underscore that word distinction, as chairman of the Co-ordinating Committee of the President's Conference on Industrial Safety.

Bill Connolly probably knows more about the problems incident to the administration of workmen's compensation laws than any of us in this room. Over and beyond that, he is a grand person and I know that his remarks this afternoon will go directly to the basic issues that are involved in this discussion.

BY MR. CONNOLLY:

(Mr. Connolly read a prepared paper which is on file at the Saranac Laboratory).

BY MR. WATERS:

Thank you, Bill, that was beautifully done.

Our next participant is so well known to this audience that I will make but brief comment in his introduction. His friendship with the Saranac Laboratory, for industry,

Statement by William L. Connolly, Director
Bureau of Labor Standards, U. S. Department
of Labor

Seventh Saranac Symposium, Saranac Lake, N.Y.
Friday September 26, 1952.

SUBJECT: — Administration of Workmen's Compensation Laws
as They Relate to the Pneumoconioses

I accepted Ted Waters' invitation to participate in this panel discussion with a great deal of personal pleasure and official satisfaction.

All of us, and the many American workers who are exposed to, or suffering from the effects of, ^{lung disease} pneumoconiosis, are grateful to the Saranac Laboratory for this opportunity to review and to evaluate our experiences in the various medical, legal, preventive and administrative phases of this problem.

The personal pleasure, which I share with all of you, arises from a natural concern with the welfare of my country's wage earners, and stems in large degree from my background as a union and a governmental labor official, whose principal preoccupation has been with that welfare.

My official satisfaction rests upon the fact that the Bureau of Labor Standards, which I now head, was the instrument used by the Secretary of Labor ^{some} 16 years ago in assembling, organizing and operating the National Silicosis Conference.

Most of what has been done since that time in the field of silicosis and the ^{and diverse} other pneumoconioses has been guided by the general principles, ^{as} laid down by that Conference. And this conference is, in a manner of speaking, a continuation of that earlier assembly.

As many of you know, the Bureau of Labor Standards is a service, rather than an operating, agency. We enforce no laws. By ourselves, we administer no legislative programs, we formulate no standards.

It is our purpose to provide technical and consultative services to others

In formulating standards and in devising and improving administrative methods and techniques in the fields of labor, social and related legislative and promotional programs.

We operate, on behalf of the Secretary of Labor and the President of the United States, continuing conferences in such fields as labor legislation, industrial safety, employment of the physically handicapped, and child labor and youth employment. These are supplemented by special conferences like the one on silicosis.

We participate in the affairs of a number of other organizations, such as the International Association of Governmental Labor Officials, the International Association of Industrial Accident Boards and Commissions, in which the Bureau functions as a secretariat, and in such others as the National Safety Council and the American Standards Association.

In all of these, we make available to the group and its individual members the assistance of our full-time specialists, both in helping to agree upon generally acceptable standards and in putting these standards into effect at the operating level.

This, we feel, is a proper Federal service - to assist the States, which make up our federated system of government, and groups of the citizens who make up our democracy, to devise proper standards and objectives for their efforts, and effect methods of attaining them.

In my remarks here today, I intend to adhere to the familiar role of my Bureau - of keeping the principles and objectives clearly in mind, and of passing along such information as we have gathered concerning the progress made toward their realization.

The National Silicosis Conference was composed of four major committees - one on prevention through medical control; another on prevention through engineering

control; a third on the economic, legal and insurance phases of the silicosis problem; and one on its regulatory and administrative phases.

The principles and objects with which we on this panel are concerned were outlined by the last committee, that on regulatory and administrative phases.

I believe we can all agree with the first principle stated by that committee in its report - that "the primary solution of the silicosis problem is prevention of the disease." Injury prevention is inherent in workmen's compensation. And any system of law or administration which seeks to minimize costs to employers by low benefits or by undue restrictions on the rights of claimants, is derogatory both of the justice of the system to injured workers and to its effectiveness in stimulating safety.

The committee also recommended "disability or death due to silicosis and other occupational dust diseases" should be compensable, that the coverage be compulsory rather than elective, and that disabled employees are "entitled to the same amount of compensation as those disabled as a result of accidental injury."

As its third principle the committee recommended the making of dismissal payments to employees separated from their jobs because of silicosis, for rehabilitation purposes.

The three major recommendations of the committee were then - ⁽¹⁾ prevention, ⁽²⁾ just compensation, and ⁽³⁾ rehabilitation.

There were other recommendations, too.

Recognizing the problem of accumulated exposure the committee recommended a sliding scale of benefits payable by the employer starting with 50 percent of the statutory benefits for accidental injury, and increasing to 100 percent within a period of 5 to 7 years, the difference being paid to the disabled employee out of a special fund established by the State.

The majority of the committee recommended the establishment of a medical board of experts on dust diseases, whose findings on questions of medical fact should be final. A minority report, ^{however,} filed by labor members of the committee, strenuously objected to the recommendation that the board's findings be final.

Concerning time limitations on claim filing, the committee recommended that "there should be not less than a 2-year period for filing claims subsequent to the date of last exposure."

The committee recommended medical care "up to that point where, in the judgment of the medical board, it is manifestly of no material help to the claimant."

That is where we started about 16 years ago.

Now - how far have we come since then?

There can be no doubt of the tremendous progress made in the field of prevention. The span in time from Gauley Bridge in West Virginia, to New York's Delaware Aqueduct was short; in terms of progress it was generations.

In some of the other fields covered by committee recommendations, the picture is not so bright. And experience has opened up several of the committee recommendations to question.

Twenty-six States, Alaska, the District of Columbia, Hawaii, and the Federal government provide compensation for all occupational diseases. Eighteen States and Puerto Rico cover only listed diseases.

In 21 States, benefits payable for silicosis and asbestosis are subject to special limitations. In 16 States no benefits are paid for partial disability. One State provides no allowances for medical care in the case of silicosis.

Thus, we still have far to go in according to victims of ^{dust diseases} pneumoconioses the same treatment accorded to workers suffering from accidental injury or other occupational diseases.

The continued use of schedules to cover occupational diseases is also a *very grave* problem. For example, beryllium is covered in only two of the 13 States which compensate for listed diseases only. The legislative process is slower than science, and the coverage of occupational diseases by listing in the law is bound to work injustice and hardship.

Vesting in medical boards the power to make final findings of fact in medical questions - as originally recommended by the National Silicosis Conference - has had serious drawbacks, ^{from} constitutional and administrative viewpoints.

Experience has indicated that the prevalent practice of letting the time limitation on claim filing run from the date of last exposure works serious injustice on persons suffering the delayed effects of many occupational diseases.

These are a few of the more important subjects in connection with our problem which need exploration. In our deliberations we should keep before us the primary objectives of prevention, justice and rehabilitation, outlined by the National Silicosis Conference. All other things - including immediate financial impact on employers, and problems for administrators, must be subordinated to these main goals.

U. S. DEPARTMENT OF LABOR
BUREAU OF LABOR STANDARDS
WASHINGTON

September 3, 1952

Dr. Arthur J. Vorwald, Director
The Trudeau Foundation and
The Saranac Laboratory
P. O. Box 551
Saranac Lake, New York

Dear Dr. Vorwald:

Thank you for sending a copy of the printed program for the Seventh Saranac Symposium. The schedule is completely in line with my understandings with Mr. Waters.

An advance copy of my statement at the opening of the panel discussion is enclosed in compliance with your request. Another copy has been sent to Mr. Waters, along with the questions which I am suggesting.

Since my participation in the Symposium is strictly "in line of duty," my expenses will be paid out of our travel appropriation.

I am looking forward to being with you later this month.

Very truly yours,

WILLIAM L. CONNOLLY
Director

BY MR. WATERS:

Thank you, Bill, that was beautifully done.

Our next participant is so well known to this audience that I will make but brief comment in his introduction. His friendship with the Saranac Laboratory, for industry,

for management and labor and for the successful administration of compensation laws is so well known to each and every one of us that it is not necessary for me to comment upon them. It has been my privilege to have known him - to have known many insurance executives during the course of my professional practice, but I know of no one, and I say this in all sincerity and honesty, I know of no one who has a better understanding of the rules and responsibility of insurance carriers in the field of workmen's compensation than Ben Keuchle.

He was schooled in the office of the Industrial Commission of Wisconsin. I happened to have lunch with him today and I said, "Ben, just how many years have you been wrestling with this problem?" He said, "Ted, I began in 1912," so he has had forty years at the task. He later became and now is Vice-President and Claims Manager of the Employers Mutual Liability Insurance Company of Wisconsin. This afternoon, he brings to us a healthy, helpful and progressive viewpoint with respect to the responsibility of insurance in the administration of compensation laws. Mr. Keuchle.

BY MR. KEUCHLE:

(Mr. Keuchle read a prepared paper, which is on file with the Saranac Laboratory).

BY MR. WATERS:

EMPLOYERS MUTUAL LIABILITY INSURANCE COMPANY OF WISCONSIN
EMPLOYERS MUTUAL FIRE INSURANCE COMPANY
WAUSAU, WISCONSIN

B. E. KUECHLE
VICE-PRESIDENT AND CLAIM MANAGER

August 26, 1952

Arthur J. Vorwald, M. D., Director
Edward L. Trudeau Foundation
Trudeau, New York

I am attaching a copy, not completely polished, of my remarks for the Symposium. Look it over, please, and feel perfectly free to use your red pencil.

I'm merely sending you this memo now so that you may know I'm working on the paper. I'm sending it to several people in Wisconsin and elsewhere for comments, to be sure I have all my information correct. Prior to the session I'll send you a final draft.



BEKuechle 22

Arthur J. Vorwald, M. D., Director
Edward L. Trudeau Foundation
Trudeau, New York

Rec'd 9/13/52
Address reply to:



Home Office
Wausau, Wisconsin

August 29, 1952

Mr. T. C. Waters
Mullikin, Stockbridge and Waters
10 Light Street
Baltimore 2, Maryland

Dear Ted:

I'm sending you what will probably be the final draft of my remarks to be made at Saranac, unless you and Art Vorwald have some additional suggestions. Art is getting a copy of this letter.

I have had the paper looked over by Mr. Kenneth Grubb of Milwaukee, who is probably the outstanding Wisconsin attorney in connection with silicosis problems, and by Mr. Harry Nelson, Director of Workmen's Compensation of the Industrial Commission. In returning the draft of the talk, Mr. Nelson made this comment in the last paragraph of his letter: "As I read your talk, I again wondered why other states flounder around with elaborate and abstruse provisions for partial disability which is really nonexistent."

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ADMINISTRATION OF WORKMEN'S COMPENSATION LAWS AS THEY RELATE
TO THE PNEUMOCONIOSES*

by

B. E. Kuechle - Vice President - Employers Mutual
Liability Insurance Company of Wisconsin

In Wisconsin we have lived with a so-called occupational disease law since July 5, 1919. On that date an amendment to the Wisconsin Law became effective which said that the provisions of the compensation law "are extended so as to include, in addition to accidental injuries, all other injuries, including occupational diseases, growing out of and incidental to the employment." At present our law defines an injury as mental or physical harm caused by accident or disease.

Under this amendment, employers have been required to pay, under the definition of injuries, claims arising out of disabilities due not only to the common occupational diseases such as dermatitis, silicosis, and asbestosis, but claims arising from disabilities caused by smallpox, measles, scarlet fever, and other similar nonindustrial diseases, when the facts indicated that these diseases were contracted through industrial exposure, as for example, a teacher contracting measles from an epidemic of this disease in her schoolroom.

Since 1919 there has never been an attempt through the legislature to restrict the phraseology of the amendment I quoted except in one instance. This occurred during the days of the nationwide silicosis scare when many employees who had been exposed to silica, and discharged because of the depression attempted to collect for either a real but usually an imaginary pulmonary disability. In one of our granite-cutting centers, work practically ceased because of competition from Minnesota, which had no occupational disease law. As a result, Minnesota manufacturers were able to export stones, both structural and monumental, into Wisconsin at a great deal lower cost than they could be processed in the state. The Union requested the legislature to reduce benefits under the compensation law for silicosis claims by 50 per cent. The bill was passed by both houses of the legislature, but was vetoed by the Governor.

* Delivered at Seventh Saranac Symposium ** Saranac Lake, New York, on September 26, 1952.

This incident awakened interested parties in Wisconsin, including employers, labor, and the Industrial Commission to the fact that an occupational disease law such as ours had to be wisely enacted and administered so that compensation could be liberally granted to employees actually disabled with a pulmonary disease and its complication of tuberculosis and still not throw out of employment individuals with minimal disease with no complicating infection who were not subjected to excessive exposure which would cause progression of their disease.

The steps taken to accomplish these ends, while not discussed in chronological order, were the following:

It was recognized that merely because an individual might have fibrosis, even approaching the nodular stage, as for instance in silicosis, did not necessarily mean that that employee would be better off either socially or economically to be forced to leave his trade, particularly where dust content under control measures was within reasonably safe limits even though he might be paid a small amount of compensation for a so-called medical partial disability. This principle was recognized by the legislature and presently the Commission has the authority to award compensation up to \$3500 where such a medical disability exists, and it is inadvisable for the employee to continue working under further dangerous exposure. Should an employer discharge an employee because of a nondisabling fibrosis prior to a finding by the Industrial Commission that it is inadvisable for the employee to continue employment, the employer who so discharges his employee shall be primarily liable for this allowance even though insured for his compensation liability. This payment up to \$3500 is in the nature of a rehabilitation allowance. If subsequently, the employee should actually become disabled, he is not barred from making a further claim although any allowance granted him for the nondisabling condition shall be applied against the award for the disabling condition.

I have in my files a letter from Mr. O. A. Fried, Chief Statistician of the Wisconsin Industrial Commission, reading as follows: "I have checked back to 1940 before running across a single case of 'nondisabling silicosis.' In the single case the injured was awarded \$655 indemnity."

It was further recognized early in the administration of the law that tuberculosis as a complication of fibrosis was the principal factor in producing disability and that it was important to eliminate contact infection. Also that certain individuals seemed to have a more rapid progression of fibrosis even where the exposure was within reasonably safe limits, and that, therefore, removal from further exposure was in the interest of such a worker.

Under the Wisconsin Law the Industrial Commission has the authority to issue orders which have the effect of law, to provide for the safety and health of employees. While no formal orders have been issued requiring physical examinations of employees, our Industrial Commission did sponsor several conferences of representatives of labor and industry with the idea, as stated in the call of one of these conferences, "to endeavor to work out some uniform physical examination program which can be submitted to organized labor and which should have their unqualified approval."

Such a program was approved by representatives of labor and industry, under the title of "Physical Examinations of Industrial Workers" and published by the Wisconsin Industrial Commission in 1939. The statutes are sufficiently adequate to assure against any danger of abuse by industry of the physical examination program as recommended by the Industrial Commission, and I know of no instance where any penalties have been applied against any employer.

To add further incentive for proper measures of dust control, the Industrial Commission has the authority to penalize an employer 15 per cent of the primary compensation and it is not permissible to insure this penalty liability.

Another problem in administration which had to be resolved was the question of prorating liability, both as between employers and as between insurance carriers. In the
(1)
Schaefer case the court held that where an individual was simultaneously employed by three employers who all contributed to the exposure, the liability should be prorated among the three employers employing the injured at the time the disability began.

(1) Schaefer and Company v. Industrial Commission, 195 Wis. 317.

However, the court denied the contention of these three employers that two earlier employers should also be held liable. This ruling was subsequently strengthened by an amendment to the law which fixes the date of disability and the liability for the disability on "the last day of work for the last employer who caused the disability." While the Industrial Commission is not bound by any specific method in the law as to determination of whether a certain period of employment caused the disability, it has generally been held by the Commission that approximately 90 days of exposure for the last employer would be sufficient to spell liability. An employer who is held responsible under these conditions may not ask for contribution from previous employers even though the exposure may have been of a massive character prior to the last employment and minimal during the period of employment for the last employer.

The insurer of the employer on the last day of work of the employee assumes the full responsibility. Our Supreme Court held that the relative length of exposure is not important and that any other rule would make it very difficult to administer the occupational disease provisions of the act. The Court said that an insurance carrier held on such claim would not be materially harmed because under the law of averages such burdens would be equalized between carriers.

The Wisconsin statute is designed to protect an employee for disability that may occur even after exposure has ceased. Lack of notice of injury is not a bar to recovery if the employer was not misled. Regardless of whether notice was received by the employer or not, the claim does not become outlawed until two years from the date of injury or death or from the date the employee or his dependents knew or ought to have known the nature of the disability and its relation to the employment. There is another saving provision which holds that the right to compensation shall not be barred if the employer knew or should have known within the two-year period that the employee had sustained the injury on which the claim is based. In cases of rehabilitation awards for cases of non disabling silicosis, if disability or death eventually ensue, an application for benefits may be filed at any time. In all cases, the general six-year statute of

There have been a number of cases in Wisconsin where employees who had been retired on pension eventually became disabled or died from silico-tuberculosis. Full compensation was awarded either to them or their dependents.

That the Wisconsin Plan has proven very satisfactory, is evidenced by a comparison of compensation insurance rates in two typical industrial classifications having a known silicosis hazard. Our actuarial department has supplied me with the rates per \$100 of payroll for these two classifications in 1928, 1934 (which was the height of the silicosis epidemic) and 1952. Since 1928, benefits under the Wisconsin law have been increased by successive legislatures by 56 per cent.

| <u>Code</u> | <u>1928</u> | <u>1934</u> | <u>1952</u> |
|---|-------------|-------------|-------------|
| 1803 - Stone Cutting (not otherwise classified) | 3.74 | 10.98 | |
| 1807 - Stone Cutting - Less than 5% Silica | | | 2.57 |
| 1808 - Stone Cutting - 5% or more Silica | | | 7.43 |
| 1082 - Steel Castings | 2.55 | 8.06 | 2.54 |

The classification for stone cutting, not otherwise classified, was discontinued in the Insurance Manual some time after 1928.

Comment

MR. WATERS: Thank you, Bill, for an excellent presentation.

Our next participant is so well known.....

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.....to this subject.

Remarks by B. E. Kuechle

In Wisconsin we have lived.....

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..... some time after 1928.

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*This is a tentative copy
See office version*

ADMINISTRATION OF WORKMEN'S COMPENSATION LAWS AS THEY RELATE
TO THE PNEUMOCONIOSES

In Wisconsin we have lived with a so-called occupational disease law since July 5, 1919. On that date an amendment to the Wisconsin Law became effective which said that the provisions of the compensation law "are extended so as to include, in addition to accidental injuries, all other injuries, including occupational diseases, growing out of and incidental to the employment." *insert*

Under this amendment, employers have been required to pay, under the definition of injuries, claims arising out of disabilities due to not only the common occupational diseases such as dermatitis, silicosis, and asbestosis, but claims arising from disabilities caused by smallpox, measles, scarlet fever, and other similar nonindustrial diseases, when the facts indicated that those diseases were contracted through industrial exposure, as for an example, a teacher contracting *small pox* measles from an epidemic of this disease in her schoolroom.

Since 1919 there has never been an attempt through the legislature to restrict the phraseology of the amendment I quoted except in one instance. This occurred during the days of the nationwide silicosis scare when *many* every employee who had been exposed to silica, when thrown out of work because of the depression attempted to collect for either a real but usually an imaginary pulmonary disability. In one of our granite-cutting centers, work practically ceased because of competition from Minnesota, which had no occupational disease law. As a result, Minnesota manufacturers were able to export stones, both structural and monumental, into Wisconsin at a great deal lower cost than they could be processed in the state. The Union requested the legislature to reduce benefits under the compensation law for silicosis claims by 50 per cent. The bill was passed by both houses of the legislature, but was vetoed by the Governor.

This incident awakened interested parties in Wisconsin, including employers, labor, and the Industrial Commission to the fact that an occupational disease law such as we

had on our books had to be wisely administered so that compensation could be liberally granted to employees actually disabled with a pulmonary disease and its complication of tuberculosis and still not throw out of employment individuals with minimal disease with no complicating infection who were not subjected to excessive exposure which would cause progression of their disease.

The steps taken to accomplish these ends, while not discussed in chronological order, were the following:

It was recognized that merely because an individual might have fibrosis, even approaching the nodular stage, as for instance in silicosis, did not necessarily mean that that employee would be better off economically to be forced to leave his trade, particularly where dust control measures were within reasonably safe limits even though he might be paid a small amount for a so-called partial medical disability. This principle was recognized by the legislature and presently the Commission has the authority to award compensation to an individual up to \$3500 where such a medical disability exists, where it is inadvisable for the employee to continue working under further dangerous exposure. If an employer discharges an employee because of such a nondisabling fibrosis prior to a finding by the Industrial Commission that it is inadvisable for the employee to continue employment, the employer who so discharges his employee shall be primarily liable for this allowance even though insured for his compensation liability. This payment up to \$3500 is in the nature of a rehabilitation allowance. If subsequently, however, the employee should actually become disabled, he is not barred from making a further claim although any allowance granted him for the nondisabling condition shall be applied against the award for the disabling condition.

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complication of fibrosis was the principal factor in producing disability and that it was important to eliminate contact infection. It was also recognized that certain individuals seemed to have a more rapid progression of fibrosis even where the exposure was within reasonably safe limits, and that, therefore, removal from further exposure was in the interest of the worker.

Under the Wisconsin Statutes the Industrial Commission has the authority to issue orders which have the effect of law to provide for the safety and health of employees. While no formal orders have been issued requiring physical examinations of employees, our Industrial Commission did sponsor several conferences of representatives of labor and industry with the idea, as stated in the call of one of these conferences, "to endeavor to work out some uniform physical examination program which can be submitted to organized labor and which should have their unqualified approval."

A program was approved by representatives of labor and industry, under the title of "Physical Examinations of Industrial Workers" and published by the Wisconsin Industrial Commission in 1939. The statutes are sufficiently adequate to assure against any danger of abuse by industry of the physical examination program as recommended by the Industrial Commission, and I know of no instance where any penalties have been applied against any employer.

To add further incentive for proper measures of dust control, the Industrial Commission has the authority to penalize an employer 15 per cent of the primary compensation and it is not permissible to insure this penalty liability.

Another problem in administration which had to be resolved was the question of prorating liability, both as between employers and as between insurance carriers. In the

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Schaefer case the court held that where an individual was simultaneously employed by three employers who all contributed to the exposure, the liability should be prorated among the three employers employing the injured at the time the disability became effective. However, the court denied the contention of these three employers that two earlier employers should also be held liable. This ruling was subsequently strengthened

(1) Schaefer and Company v. Industrial Commission, 185 Wis. 317.

by an amendment to the law which fixes the date of disability and the liability for the disability on "the last day of work for the last employer who caused the disability."

While the Industrial Commission is not bound by any specific phraseology in the law to determine whether employment caused the disability, it has generally been held by the Commission that approximately 90 days of exposure for the last employer would be sufficient to spell liability. An employer who is held responsible under these conditions may not ask for contribution from previous employers even though the exposure may have been of a massive character prior to the last employment and minimal during the period of employment for the last employer.

The insurer of the employer on the last day of work of the employee assumes the full responsibility. Our Supreme Court held that the relative length of exposure is not important and that any other rule would make it very difficult to administer the occupational disease provisions of the Act. The Court held that an insurance carrier held on such claim would not be materially harmed because under the law of averages such burdens would be equalized between carriers.

The Wisconsin statute is designed to protect an employee for disability that may occur even after exposure has ceased. Lack of notice of injury is not a bar to recovery if the employer was not misled. Regardless of whether notice was received by the employer or not, the claim does not become outlived until two years from the date of injury or death or from the date the employee or his dependents knew or ought to have known the nature of the disability and its relation to the employment. There is another saving provision which holds that the right to compensation shall not be barred if the employer knew or should have known within the two-year period that the employee had sustained the injury on which the claim is based. In cases of rehabilitation awards for cases of disabling silicosis, if disability or death eventually ensue, an application for disability or death benefits may be filed at any time. In all other cases, the general six-year statute of limitations applies.

There have been a number of cases in Wisconsin where employees who had been retired

on pension and who eventually became disabled or died from silico-tuberculosis had full compensation awarded either to themselves or their dependents.

That the Wisconsin Plan has proven very satisfactory, is evidenced by a comparison of compensation insurance rates in two typical industrial classifications having a known silicosis hazard. Our actuarial department has supplied me with the rates per \$100 of payroll for these two classifications in 1928, 1934 (which was the height of the silicosis epidemic) and 1952. Since 1928, benefits under the Wisconsin law have been increased by successive legislatures by 56 per cent.

| <u>Code</u> | <u>Rate</u> | | |
|---|-------------|-------------|-------------|
| | <u>1928</u> | <u>1934</u> | <u>1952</u> |
| 1803 - Stone Cutting (not otherwise classified) | 3.74 | 10.98 | |
| 1807 - Stone Cutting - Less than 5% Silica | | | 2.57 |
| 1808 - Stone Cutting - 5% or more Silica | | | 7.43 |
| 3082 - Steel Castings Foundries | 2.55 | 8.06 | 2.54 |

The classification for stone cutting, not otherwise classified, was discontinued in the Insurance Manual some time after 1928.

Comment

(MR. WATERS: Well done, Ben. More power to you and to your insurance associates in this particular problem.

I regret that Mr. Earle T. Andrews, who was scheduled to appear on the program and intended to participate in this session as one representing the viewpoint

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of management, is unable to be here. We are fortunate, however, in having found a substitute who will more than fill the job. Mr. Otto A. Wisely, Chairman of the Commission of the State of Utah administering the compensation law of that state, has served in that capacity for the past eleven years and is the father of the occupational disease law, with particular reference to the pneumoconioses, which became effective during those years. He is well-informed on the whole subject of labor and industrial relations and will speak on the responsibility of management in the administration of the law and will deal with such subjects as prevention and rehabilitation.

By Mr. Waters:

Well done, Ben. Ben's remark on confusion reminds me of a story on one of my professional associates which I am going to take the liberty to tell. It's very short. He defended a criminal convicted upon a criminal charge and sentenced to the electric chair. Just prior to the execution, he visited the death house. Before leaving the prisoner, he tried to say something that he thought would be comforting and stumbled around in his mind for the proper expression and found that he could not say, "I hope to see you soon", because he did not hope to see him soon where he was going, nor could he say, "Happy Landing", because he knew that his landing would not be happy. Like a flash, the top expression came to him, and he left him with the greeting "More power to you", so Ben, I say to you and to your insurance associates, "More power to you in this particular problem".

It is with regret that I must tell you that Mr. Earle T. Andrews, who was on the program and had intended to participate in the session this afternoon, representing the point of management, is unable to be here. He called me last night. His company is faced with a strike on Monday morning, and he asked me to convey to Doctor Vorwald and to the audience, his sincere and deep regret that he could not come.

However, I know that we are all most fortunate in that we have found a substitute who will more than fill the

job. Mr. Otto A. Wisely, is the Chairman of the Commission of the State of Utah, administering the compensation law of that state. He is a graduate of the Law School of the University of Utah and is a clinical lecturer in that university in its medical college. He is Chairman of the Commission and has so served for the period of the last eleven years. During the time that the occupational disease law with particular reference to the pneumoconioses became effective, and he has been its daddy.

He is well informed on the whole subject of labor and industrial relations. Perhaps I should express one personal criticism of him, because on more than one occasion, he has had to serve the role of a character witness for our good friend, Paul Richards from the same state. I have asked him to say just a word, particularly with respect to the responsibility of management in the administration of the law, dealing with such subjects as prevention and rehabilitation. Commissioner Wisely.

BY COMMISSIONER WISELY:

Thank you, Ted. I am very happy that I have been able to attend this symposium. I came here because of the insistence of Doctor Paul Richards, and that insistence was in the nature of a telephone call to Governor Fee telling him, you tell Otto to goto Saranac Lake. I told the Governor I wouldn't do it. He knew that - he's a very good friend

BY COMMISSIONER WISELY:

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Comment

MR. WATERS: That was a well-presented and very effective statement of
Mr. Hill's point of view. On many of the issues I happen to disagree with
Mr. Hill but that is my privilege and the similar privilege of those in the
audience.

The next speaker, William L. Connolly, is a
.....
.....involved in this discussion.

Remarks by William L. Connolly

I accepted Ted Waters' invitation to participate in this.....
.....
.....to these main goals.

shall we
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Page as
submitted OK
Connolly

June 20
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Wisely

Remarks by Otto A. Wisely

I am happy that I have been able.....
.....
.....we'll see that they do.

Paper by Otto A. Wiesley

The viewpoint of management is best evidenced by acts rather than words. I shall try to show you that in Utah management's attitude is excellent. Please bear in mind that Utah is a small state and that our methods may not fit your situation.

Since 1945 labor and management have cooperated in legislative programs to the end that all legislation pertaining to workmen's compensation and occupational disease has been agreed upon in advance of the introduction of bills. No changes have been made by the legislature after the bills were submitted. The Utah Occupational Disease Disability Law (1941) is the result of the labors of the Commission and a joint labor-management-medical profession committee. We now have a standing study committee. The importance of this procedure is that it eliminates litigation and argument. Both sides must and do admit that although we do not like some features of the law, nevertheless, it is our law and we will abide with it in good spirit.

The number of contested cases is indicative of the attitude of management, particularly in view of the fact that management is on the losing end in the majority of cases contested. We receive thirty thousand accident reports annually. Last year only seventy-eight went to formal hearing. Of this number only two involved an occupational disease. During the last four years only nine cases were appealed to our Supreme Court. One of these involved an occupational disease - silicosis.

Approximately seventy-five cases have been processed by our Medical Panel. As heretofore stated, only one case appealed to the Supreme Court and only two cases were formally heard by the Commission. Both labor and management accept the medical findings and ratings of the Panel without question.

Disability rating is a problem. We all realize that we cannot rate with mathematical accuracy. In the absence of infection we usually rate silicotics as follows: S1 - 0%; S2 - 25-50%; S3 - 50-100%. Sixty-five percent and up is usually considered total. If infection is present, then the rating is temporary and total in all cases.

Management believes in speedy disposition of cases because of the psychological effect on the injured man and maintenance of good labor-management relations. Contested cases are speedily set for hearing; hearings are brief; delays are not countenanced and decisions are quickly released.

Management believes in giving the injured man the best possible medical care. For all practical purposes our law places no dollar limit on medical care. Hospitalization is, however, limited to \$1,500.00 in occupational disease cases. Management has taken the lead in efforts to increase medical benefits.

Both labor and management insist on honest, scientific medical testimony. The Utah State Medical Society has cooperated at all times and in every

way possible to eliminate the fee doctor and the medical advocate from industrial medicine and particularly from formal hearings. If we draw a bad number we send the transcript to the Board of Censors and we get results.

Neither labor nor management, and certainly not the Commission, believe in excessive legal fees. In fact, fees are very low. Beneficial legislation demands low fees. Our ceiling is ten percent. The law is intended to benefit the employee and his dependents and not the legal profession. Legal fees, we discovered, have a direct bearing on the number of cases contested.

Another important development credited to both labor and management is the compulsory autopsy law adopted in 1949. If compensation is claimed, the Commission, on its own motion, or on request of any interested party, may order an autopsy. If the widow or dependents refuse permission, no compensation is payable. Twenty autopsies have quite positively confirmed the findings and diagnoses of our Medical Panel. This, more than any other factor, has instilled confidence in the minds of all concerned that the panel method really can work.

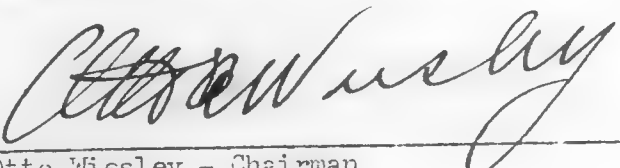
Management's viewpoint is illustrated by the fact that since 1941, effective date of the occupational disease law, management has not taken advantage of the many defenses available. Our law defines disablement as inability to carry on any occupation for remuneration or profit. This defense has never been used although in many cases it was a complete defense. Dust counts are no longer requested. Length of exposure, unless obviously absurd, is not questioned. Of course, when two or more employers are involved, the thirty day and last employer requirement becomes important as between employers but not as to the employee.

In short, in Utah, occupational disease claims, especially silicosis, are boiled down to the simple fact of employment, the existence of the disease, disablement- medically, and in a few cases - statute of limitations. The metal mining industry has recognized and assumed a moral obligation for silicotics. The contest, if any, is between employers.

Finally, management has cooperated wholeheartedly in the open door policy of the Commission. We have no star chamber sessions, no inner sanctums. Whenever possible we resort to a sort of pre-trial discussion which almost always obviates a hearing.

If we succeed in solving the job of rehabilitation and job placement, Utah will have solved its major occupational disease problems. Both labor and management are working on the problem.

Thank you.


Otto Wiesley - Chairman

INDUSTRIAL COMMISSION OF UTAH and
LABOR RELATIONS BOARD.

Dated: July 6, 1953

Comment

MR. WATERS: It is a wonderful experience when you can hear a Commissioner tell that type of story.

I do not have to remind this audience of the responsibility and role of the Medical Director of our large corporations. The physician who is successful in that role is more than a doctor - he enjoys a personal relationship with the men subject to his charge and is closer than any other representative of management. He becomes father confessor to the company employees - he is successful only in so far as he instills complete confidence of his men in the discharge of his duties.

Dr. Lloyd Hamlin is the Medical Director of one of the largest and best managed of our industrial corporations, the American Brake Shoe Company. His

experience has been so extensive that he is fully familiar with the medical problems presented by pneumoconiosis, the role of Medical Departments and their relation in the administration of workmen's compensation laws.

Wisley

Chapter Thirty-^{three}~~two~~

Administration of Workmen's Compensation Laws as They Relate
to the Pneumoconioses

The administration of workmen's compensation laws as they relate to the
pneumoconioses was discussed by a panel having the following members:

Moderator : Theodore C. Waters

Panel members : James ^{L.}L.Hill

William L. Connolly

B. E. Kuechle

Otto A. ^{Wisley}Wisely

Lloyd E. Hamlin, M.D.

Introductory remarks by Theodore C. Waters

The panel discussion that will be

.....

.....will be appreciated.

The first speaker on the program.....

.....

.....and its administration.

Omit
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SARANAC SYMPOSIUM
ON "PNEUMONIC CONIUSES"

SARANAC LABORATORY
Saranac Lake, New York

September 22-26, 1952

Panel on
"Administration of Workmen's Compensation Laws"

INTRODUCTORY REMARKS

The Panel discussion that will be presented at this session deals with the "Administration of the Workmen's Compensation Laws" of the several states relating to "pneumoconioses". The parties in interest are four: the employee, the employer, the insurance carrier and the public. Industrial workers have their principal contact with the administration of American Jurisprudence before our various compensation commissions, and the successful administration of the laws is a matter of concern to the four parties to whom I have just referred.

May I say just a word with respect to our procedure. I shall introduce each Panel participant and he will make a brief statement as to his point of view in the administration of the laws. This will be followed by the presentation of prepared questions. It is hoped that the question and answer period will promote discussion and develop the controversial aspects of the subject. Following the prepared questions, the audience will be invited to direct questions to the Panel and to participate in the discussion. No one has been told what to say or what not to say. Even though we may disagree with the point of view of one

or more of the speakers or with those in the audience, it is our desire to present a forum for the open and frank discussion of the subject. May I ask those in the audience to present their questions in as simple and direct a form as possible so they may be understood by the participants in the Panel and others in attendance. It is hoped that several of the participants in the Panel will join in the answer to the questions, but limitation of time requires imposition of a period of not more than three minutes for the answer to given questions. Your cooperation in this respect will be appreciated.

INTRODUCTION OF PARTICIPANTS
OF THE PANEL ON

"THE ADMINISTRATION OF WORKMEN'S COMPENSATION LAWS"

HONORABLE JAMES L. HILL, Commissioner
Workmen's Compensation Commission
Lansing 13, Michigan

The first speaker on the program has served with distinction as a member of the Workmen's Compensation Commission of the State of Michigan. He has rendered the people of his state a distinct public service in the administration of his office.

Compensation laws are complex and those features dealing with pneumoconioses, both as they relate to the letter of the law and its interpretation, have presented peculiar problems to those charged with their administration. The State of Michigan with its vast industry has experienced all of the problems that we have been discussing during this symposium.

James L. Hill became legal advisor to the Commission in 1936, and since that time has been serving as one of its members. He is also a past president of the International Association of Industrial Accident Boards and Commissions, and brings to us a wealth of experience in formulation of legislation and its administration.

WILLIAM L. CONNOLLY, Director
Bureau of Labor Standards
U. S. Department of Labor
Washington 25, D. C.

Our next speaker, William L. Connolly, is a Director of the Bureau of Standards of the United States Department of Labor. He was called to that post after having served for 6 years as Director of Labor for the State of Rhode Island, where he supervised the administration of the workmen's compensation laws of that state. To his honor and credit, he is former President of the Rhode Island State Federation of Labor. For some years past, he has served as Chairman of the Co-ordinating Committee of the President's Conference on Industrial Safety.

Bill Connolly knows more about labor relations and responsibility of management and labor in the matter of the administration of compensation laws than most of us in this room. He is a grand person and I know his remarks this afternoon will go directly to the basic issues involved in this discussion.

**EARLE T. ANDREWS, Vice President
in Charge of Operations
Pennsylvania Glass Sand Corporation
Hancock, West Virginia**

Management has a double responsibility in the administration of Workmen's Compensation Laws: (1) To provide reasonable and adequate compensation for industrial injuries, and (2) To effect medical and engineering methods of control for the prevention of industrial injuries.

Too many modern executives have rested comfortably in plush chairs and offices with but little real concept of the working conditions to which their employees are exposed. Earle T. Andrews, our next speaker, is an engineer who has rubbed shoulders with and worked in all places and under all circumstances to which the men under his charge are subjected. He knows what silicosis is all about and he knows of the responsibility of management for the protection of his employees against that disease.

To me it is a personal privilege to participate in this program with Mr. Andrews. He and I have been associated in business over many years and I have personal knowledge of his concern for the health and welfare of the employees of his company.

**B. E. KUECHLE, Vice President
and Claim Manager
Employers Mutual Liability
Insurance Company of Wisconsin
Wausau, Wisconsin**

Our next participant is so well known throughout our audience that I will make but brief comment in his introduction. His friendship for Saranac Laboratory, for industry, management and labor and for the successful administration of compensation laws are well known to each and everyone of us. It has been my privilege to have known many insurance executives during the course of my professional practice, but I know of no one who has a better understanding of the role and responsibility of insurance carriers in the field of workmen's compensation than Ben Kuechle.

He was schooled in the office of the Industrial Commission of Wisconsin and later became Vice President and Claim Manager of Employers Mutual Liability Insurance Company of Wisconsin. This afternoon, he brings to us a healthy, helpful and progressive viewpoint with respect to this subject.

DR. LLOYD HAMLIN, Medical Director
American Brake Shoe Company
2501 Blue Island Avenue
Chicago 8, Illinois

I do not have to remind this audience of the responsibility and role of the Medical Director of our modern corporations. The physician who is successful in that role is more than a doctor--he enjoys a personal relationship with the men subject to his charge and is closer than any other representative of management. He becomes father confessor to the company employees--he is successful only in so far as he instills complete confidence of his men in the discharge of his duties.

Dr. Lloyd Hamlin is the Medical Director of one of the largest and best managed of our industrial corporations--American Brake Shoe Company. His experience has been so extensive that he is fully familiar with the medical problems presented by pneumoconioses, the role of Medical Departments and their relation in the administration of workmen's compensation laws.

CLOSING REMARKS

It is with regret that I must bring this Panel discussion to a close. Many of the questions that have been presented are highly controversial and I am certain that many of us have differences of opinion with respect to these matters.

We are indebted to Saranac Laboratory for this opportunity to have received such authoritative information and advice about problems with which all of us must deal in the transaction of our business.

On your behalf I wish to thank the participants in the Panel for the excellent presentations that they have made and for their honest expressions of opinion; on their behalf, I wish to thank you for your interest, consideration and participation in the proceedings of this Panel. I know that Dr. Vorwald and his associates in the Laboratory are deeply indebted to you and to the members of the Panel for your joint contributions.

of mine, of course, that's the only reason I could tell him that. He said, "All right, Otto, if you don't think you could go, but I think you should go", and I'm very happy that Paul induced the Governor to order me to attend this symposium.

There is another reason why I am happy to be here. Very early after the occupational disease law was adopted, became effective, on July 1st, 1941, in Utah, my first silicosis hearing found us with very few, if any, men, medical men who knew anything about silicosis. There was one exception to that, and that's the gentleman who, this day, is still the pioneer in industrial medicine, and one of the outstanding experts in the disease of pneumoconioses, and that's Doctor Paul S. Richards, and he suggested that we had better lay a foundation, and we did.

We had Doctor Roy Gardner, Doctor George Wright, from Saranac and Doctor Hussey, who at that time, was with Johns-Hopkins. They did a splendid job, and I want to say to these men that that case, the France case, never did go to the Supreme Court. It was too damned well done, laid the foundation for all future hearings, and we're deeply indebted to these three gentlemen and to the pioneering work that Doctor Paul Richards did.

My grand daughter frequently tells me that patience is a virtue. Well, I have been patient all week. I had a

lot of questions that popped up in my mind, as the various speakers paraded across this platform, but all those questions were asked. Patience, you see, but not all of them were answered, as you well know.

Now, I have been asked to speak to you briefly on the same subject that has been discussed, from the viewpoint of management, and this will be rather a peculiar way to express the viewpoint of management, because you will not understand our philosophy and our procedures unless you come to Utah. It's easy to read a law and you may interpret it as you think any lawyer or any court would interpret it, but you'd better not pass judgment on the Utah occupational disease disability law until you come to Utah and see us in action, see what we've done.

Now, we have about 30,000 reportable injuries in the state every year, not many of them o.d.'s. Of course, and of that number last year only 78 went to a formal hearing, not a bad record, I would judge. Since we amended the statute, and created our medical panel for occupational diseases, particularly for silicosis, three and a half years ago, not one single case has gone to the Supreme Court of the State of Utah and not once did either party question the medical findings and the diagnosis of our medical panel.

The only formal hearing we have had in an occupational disease case in the last four years, posed only one

question and that was the degree of disability which, of course, was very interesting to me. I took the position that a man who is rated seventy-five percent disabled, should be rated totally disabled if, for no other reason, than that a man seventy-five percent disabled certainly is not on the labor market after that.

As far as rating his disability, we have no particular trouble. We don't contend that we're right always. We think we get within ten percent either way, generally. The parties seem to be satisfied and, after all, that's the important thing, if you make an award, is to satisfy the applicant that he has received enough money, so we get along very nicely in that respect.

Another thing about our procedure, and all the way through now, you might as well be thinking of management, as I'll show you in just a few minutes, this is management's viewpoint, we believe, in speedy action.

If the Referee -- I only have one at this time -- doesn't get the rough draft of his proposed findings of fact and conclusions of law to me within ten days after he hears a case, I'm going to get a new Referee, and that decision is out in the mail within twenty-four hours after the Referee finally sends in the final proposed findings of fact and conclusions of law.

I understand that some cases in some states have

been pending for years. If that happened in Utah. we'd get a new commission, new referee, new everything. The - it just doesn't make sense. Our procedure is extremely simple and I believe laws that we have, particularly the o.d. law and the amendments in the last few years, have the complete and always have had the complete approval of management.

As a matter of fact, I might tell you here at this point that since 1945, four successive legislative sessions, every law pertaining to labor and industry compensation, laws and labor laws, were submitted to the Senate and the House, in agreed form with a letter accompanying the bill. We don't want any amendments. We can't even cross a T or dot an i. We want those laws passed just the way we hand them to the Secretary of the Senate and the Clerk of the house, and I hope we'll succeed in doing that the next session.

Now, some of you might think that in that kind of a deal, labor would get the short end. That is not true. In every single instance, labor has received more than they requested in many, many respects, and the net result has always been a law that is completely satisfactory at the moment and later on for that matter, to both sides.

Now, as far as medical problems are concerned, we don't have any trouble with that either. If I catch a doctor who doesn't tell the truth, and some of them do not, some of them are just plain ignorant, you know, but some of

them just don't tell the truth, all we do is send the transcript of the testimony to the Utah State Medical Society and the Board of Censors makes short work of any man who gives us any ridiculous testimony.

To give you an example, if you say this wouldn't happen, it did. A man was hit in the mouth, loosened a couple of teeth and I think they were already loosened because he had a very bad case of pyorrhea, went to a dentist. A dentist pulled a couple of teeth, bled a little bit, swallowed the pyorrhea infected blood. One of our doctors took the witness stand and under oath, testified that this man developed a peptic ulcer that had reached the near perforation stage in three weeks. The pyorrhea germ did it. I tried to take him off the hook by telling him, why, what you really mean is that he had an ulcer and the germs sort of aggravated it, effected it a little bit. He didn't swallow that hook either. So I sent a record to the Medical Advisory Board. They called him in, I mean the State Medical Society. They called him in, and he said, "I didn't say it". They showed him the transcript. He said, "Well, I guess I did, shouldn't have gone quite that strong". They said, well, "Why did you say it". Well he says, "The guy told me he lived in a trailer camp and I wanted to help him". You see, there was a doctor that was practicing economy in that medicine. We have had a few like that, and as I say,

we make short work of them.

Of course, here is a secret. I'm a lawyer until recently a member of the law school faculty of the University of Utah and a lot of other civic jobs, and I want every lawyer to make a living, but if you are practicing law in Utah and you depend on workmen's compensation fees for a living, well you wouldn't even pay your office rent. Our fees are very low. There are times, however, when a lawyer will make something out of nothing, as we say, and we will pay him, order a substantial fee paid him.

Now, how do we accomplish all this? For example, the Utah Occupational Disease Disability Law has a definition. We don't like definitions in Utah. We don't have any trouble with them. We do have one definition, and that is the definition of disablement. Disablement is defined as inability to carry on any gainful work or occupation for remuneration or profit.

Well, sir, you know, for eleven years, there has not been one employer in Utah that has ever raised that defense and they could have done it in every damned case we had. Now, that's the attitude of the employer. Same thing on disability. In our state, a man must become totally disabled within two years, not from the last day of exposure, but from the last day of employment, and then he has one more year in which to file a claim. A widow has six months

in a case of death, to file a claim.

We also have in Utah, a compulsory autopsy law, for both W. C. and O. D. It has been extremely helpful and I'm getting back to this medical panel now. I'm nuts about the medical panel, in fact, so much so that I'm going to ask the next session of the legislature to amend the Workmen's Compensation Act to authorize me to appoint a medical panel to determine disputed medical questions in accidental injury cases.

We also have a non-statutory medical advisory board that examines men who claim they have a permanent partial disability following recovery and they meet on Fridays, examine these men, recommend rating to us and we always accept their rating. In eleven years I have been on the Commission, not one of those has gone to a Supreme Court and we have had less than six formal hearings growing out of that kind of procedure.

Now, you might say, well, you're either terribly dumb in Utah, employers, employees, labor unions, medical society, and so on. That isn't the case at all. We have tried to do, and we've accomplished it, I think, is to decrease, to an irreducible minimum, the distance that so frequently exists between labor and management, the commission and labor and management, the commission and the medical society, and so on. They're friends of ours.

Now, they fight like Hell over other labor problems, but when it comes to workmen's compensation, occupational disease laws, as I have already told you, they work together hand in hand in a very friendly manner and present to the legislature, the changes in the law that both parties believe, experience has taught us should come.

Now, on this question of rehabilitation, Ted, take another minute is all, we are just beginning to think about it, because our permanent partial disability law for o.d. cases has given us a little headache. That, too, I think we can solve, perhaps without any legislation. We could shoot a couple of insurance adjustors and our attorneys in Utah, there are only two out of eighty-five, and it isn't one of them, we wouldn't have any trouble.

These men who are permanently partially disabled and so rated, they go back to work in a mine if the panel says they should. They go to work anywhere in the State of Utah, but they have two insurance carriers who just raise particular Hell, and so we have not a compensation problem, no difficulty determining that they're partially disabled or what the degree of disability is, but they come back to me from Park City, the CIO Steelworkers man, he says, "Hell, Otto, the guy can't get a job; what are you going to do with him"? Well, that's the question I'm mulling around in my mind, and not long ago, and with this same employer again,

the Secretary of the Utah Miners Association, and the Secretary of the Calcium Assurers Association, and said, "Otto, when you get back, you're not going to have any more trouble with those permanent partial disability cases. The panel says they can work somewhere; we'll see that they do".

Thank you.

BY MR. WATERS:

It's a wonderful way when you can hear a Commissioner tell that type of story. I do not have to remind this audience that the responsibility and role of the medical director of our modern corporations is important. The physician who is successful in that role is more than a doctor. He enjoys a personal relationship to the man who is subject to his charge, that is probably closer than that of any other representative of management. To state it simply, he serves for the confessor to his employees, and he is successful only insofar as he instills complete confidence in his men in the discharge of his duties.

Lloyd Hamlin is Medical Director of one of the largest and best managed of our industrial corporations, the American Brake Shoe Company, a company that certainly knows all that should be known about silicosis, or the pneumoconioses. His experience has been so extensive that he is fully familiar with the medical problems presented in the administration of compensation law. Forgetting administration

of compensation laws for a moment, the problems presented by these diseases among the employees of the company and the role of the medical departments and their relationship in the proper administration of compensation laws. Doctor Hamlin.

BY DOCTOR HAMLIN:

(Doctor Hamlin read a prepared paper which is on file at the Saranac Laboratory.)

BY MR. WATERS:

I am pleased to advise the audience that the airplane schedule has been changed. The weather apparently has cleared and there is not the quite pressure of time that we were subject to when we started the meeting.

It is always the chairman - the prerogative of the chairman of any meeting to reverse his field, and I'm going to reverse my field today. There have been a number of prepared questions to be directed to the panel, but I think the audience comes first and we will welcome any questions that you may wish to direct to any individual member of the panel. In propounding those questions, kindly give your name, the company with which you are affiliated or your address and the participant of the panel to whom you wish your question directed. If you run short of questions, I've got one or two that I want to slap to the members of the panel myself, but at the moment, I invite

Remarks by Lloyd E. Hamlin, M.D.

During the month of June 1945, a man.....

.....

.....exposure to dust.

rec'd
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file

ADMINISTRATION OF WORKMENS COMPENSATION LAWS
AS THEY RELATE TO THE PNEUMOCONIOSES -
A MEDICAL DIRECTOR'S VIEWPOINT

During the month of June, 1945, a man was hired as a laborer in one of our foundries. He had previous experience as an air hammer operator and chain man in another company for a period of 5 years. His pre-placement chest x-ray revealed no evidence of silicosis or other pathological condition. Three years later in 1948, an annual chest x-ray survey conducted by our Medical Department at this location, revealed an early minimal tuberculous lesion in the upper lobe of the employee's left lung. The matter was immediately brought to his attention and he was advised to seek medical care.

There is little doubt that the disease would not have been discovered so early had it not been for the x-ray service furnished by the company as part of its regular health program. Additional independent medical studies substantiated our findings and eliminated the presence of occupational disease of the lungs. The man received sick benefits at once under the company Comprehensive Insurance Plan and shortly there-after was admitted to the city hospital. In this institution a diagnosis of silicosis was made solely on the basis of the history of employment in a foundry and a claim for compensation was filed which alleged that the disease had been acquired at our plant.

In spite of the fact that the man's entire length of service at this plant was only $3\frac{1}{2}$ years and that repeated, accurate industrial hygiene surveys during all of this time revealed dust counts far below accepted safe limits both for gross dust and free silica (average 0.3 million particles per cubic foot of air, including all inert silicates as well as free silica and iron) and in spite of the fact that expert medical testimony by exceptionally well qualified physicians definitely indicated that there was no evidence of silicosis and that the man's condition was due entirely to tuberculosis, the referee, completely ignoring the medical and legal facts submitted by the defense awarded the claimant compensation in the sum of \$4,000.

Months later after a review of the findings, the industrial commission handed down a final award of 160 weeks compensation at \$25.00 per week based on the information that the man had worked as a shakeout helper from March 18, 1946 to September 8, 1948, a period of $2\frac{1}{2}$ years. This was construed to mean that he had been exposed to "silica dust" and had thereby contracted silicosis. As had been adequately demonstrated, no unsafe exposure existed at this occupation. This fact was entirely ignored.

On September 10th, a circuit court judge affirmed the award of the industrial commission necessitating filing with the Court of Appeals where the matter now rests. In the meantime an offer of settlement from the claimant's attorney for \$3,000 has been received with the inference that a smaller sum would be considered.

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This is not by any means an unusual experience to those dealing with compensation laws nor is it necessarily true of all cases. It is recognized that there are many competent people engaged in compensation work who are doing a fine job and striving to apply the statutes in a fair and unbiased manner, but the fact remains that decisions such as the one quoted are all too frequent.

Most progressive industries today recognize the validity of giving the worker the benefit of the doubt where there is a reasonable question as to responsibility, but such obviously unfair interpretations of the Occupational Disease Laws not only arouse resentment and bitterness but in the end do not work out to the employee's advantage. Thus they do an injustice to the very ones the law is designed to help.

What effect does this have on

A. Industry

At a time when industry is becoming employee health conscious and is spending great sums of money to improve working environments and institute health and hygiene programs in a period where mounting costs and decreasing earnings might easily dictate otherwise, decisions of this sort are bound to raise the question as to whether the financial outlay is justified. In spite of opinion to the contrary all industry is not the selfish, dollars and cents wise organization it is so frequently depicted to be by those whose questionable backgrounds refute their own right to criticize. In my own company claims for total disability because of silicosis have been instituted and paid when these could have been avoided but because the moral and legal aspects were recognized, management made a sincere and honest effort to meet its just obligations.

In another instance:

A few years ago we gave employment to a man who had been working underground for 10 years in the coal mines of Pennsylvania. A preplacement chest x-ray at the time of his employment in 1945 indicated that he had rather extensive pathology in his lungs which was interpreted as silicosis with indeterminate tuberculous infection. Because industrial hygiene surveys at this plant indicated dust concentrations in 1946, 1947 and 1948 well below accepted safe limits, the man was allowed to work. He continued to be employed for a period of approximately three years at the end of which time undisputed medical evidence showed that his condition was actually better than when he began to work at the plant. Furthermore, his occupational fibrosis showed no evidence of progression whatsoever. The man's services were discontinued for "Rules Violation," following which he instituted a claim for total disability on the grounds of silicosis which he claimed was acquired at this plant.

In spite of the medical opinion of outstanding chest experts and roentgenologists and the medical opinion on behalf of the Full Board of Dust Consultants wherein all, without exception, testified positively and unequivocally that the claimant's disability was not due in any way, either directly or on the theory of aggravation to his three years work at this plant, the referee found that the claimant sustained an occupational disease due to silicosis as a result of his occupation at this plant. The case was regarded as compensable because of the "presumption" that the alleged exposure was harmful and because, as stated by the referee, there was "absence of substantial evidence to the contrary."

The case was extensively and completely tried with evidence offered on all of the issues involved, including the nature of the claimant's work, the precise exposure to which he was subjected, his condition at the beginning and termination of his employment, and on every other issue which is important in the fair evaluation and determination of an alleged silicosis claim of the type here involved.

This is not by any means an unusual experience to those dealing with compensation laws nor is it necessarily true of all cases. It is recognized that there are many competent people engaged in compensation work who are doing a fine job and striving to apply the statutes in a fair and unbiased manner, but the fact remains that decisions such as the ones quoted are all too frequent.

Management realizes its responsibility to the injured worker and cannot nor does it wish to shirk that responsibility. Healthy employees are mandatory for healthy industry. Most progressive concerns today recognize the validity of giving the worker the benefit of the doubt where there is a reasonable question as to liability, but such obviously unfair interpretations of the Occupational Disease Laws not only arouse resentment and bitterness but in the end do not work out to the employee's advantage. Thus they do an injustice to the very ones the law is designed to help.

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The right of appeal to higher courts is, of course, recognized but the very fact that so many decisions by referees and industrial commissions are reviewed and even carried to the Supreme Court is evidence that there is room for a great deal of improvement. The additional cost of such unnecessary litigation is difficult to justify and has much to do with the policy of being "fair but tough" which many industries have now been forced to adopt.

B. The Industrial Physician

The industrial physician whose first consideration should be, and usually is to the human being he is caring for, is severely handicapped by misinterpretations placed on his honest opinion, obvious disregard of qualifications and experience, and the sad but none the less actual distortion of medical facts by his own colleagues. The uninformed practitioner who becomes an expert witness on the least provocation, whether it be the hope of financial gain or a mistaken sense of generosity to an employee, the specialist or internist who has never been inside a manufacturing plant in his life but who speaks with the voice of authority on industrial diseases of the lungs, mainly from hearsay, are two of the most difficult problems with which the doctor in industry has to contend. Refusal to consider what is obviously authoritative medical opinion in preference to inexperienced and patently biased testimony on the grounds that the board or referee has no way of determining medical facts, and that as far as it or he is concerned "one doctor's opinion is as good as another's" is not a logical or honest concept of justice. Anyone who holds such a responsible position certainly

ought to possess the ability to make some distinction on the grounds of common sense even if he knows nothing of the medical terminology involved. The medical witness who deliberately distorts the truth to make his testimony favorable to either the claimant or management is just as guilty. The industrial physician is not expected to do other than render an honest opinion on a given case. No industry should attempt to influence his decision in its behalf nor should the physician allow this to happen. Management should look to the doctor, and usually does, for guidance in the disposition of any case from a medical or physical disability standpoint. When the doctor departs from these principles, he should no longer be engaged in the practice of industrial medicine.

G. Rehabilitation

Little needs to be said concerning the effect of such injudicious interpretations of the Occupational Disease Laws on rehabilitation. The individual who suffers most of course is the afflicted worker himself. Most enlightened industries realize they have an obligation to accept their share of crippled workmen, provided they are employable but when penalized by awards for compensation on the basis of alleged aggravation of previous pulmonary occupational disease or last employer responsibility, they cannot be blamed for refusing to hire such applicants. The doctor's work is made more difficult since he would like to pass these people for work but it is his duty to protect the interests of his employer as well as those of the employee.

While the foregoing remarks may apply to compensable cases generally, they are especially applicable to the pneumoconioses. The diversity of exposures and the wide variety of unrelated chest roentgenographic patterns that simulate disabling or potentially disabling occupational fibrosis of the lungs, demand that as in any other field, medical opinion should be limited to those who have adequate first hand knowledge of these conditions.

As an industrial physician who has observed this scene over a considerable number of years with a sincere human interest, I cannot help but feel there is great need for less selfishness, more tolerance, more mutual understanding and just plain honest dealing by everyone concerned in the administration and application of the compensation laws.

(Slides demonstrating variety of unrelated chest roentgenographic patterns which may be and frequently are mistaken for silicosis.)

QUESTION I.

What should the physician's attitude be toward the company and employees he serves when dealing with compensation claims for pulmonary occupational disease?

- A. (1) First of all he must decide whether in his opinion the claim is legitimate as far as the employee's physical status is concerned.
- (2) When convinced that this is so, he should make every effort to see that the employee gets due consideration. It is his duty to do this, and to demonstrate to management that it must accept its proper responsibility. He must do this even in the face of adverse pressure from his employer where he might be expected to render an opinion favorable to his (the employer's) side of the case. The doctor should stand on this principle when he knows he is right and fight for it and the employee even to the extent of losing his own position.
- (3) On the other hand, he should be equally considerate of the company and should do all he can to protect its interests when he is convinced that the employee's claim is not legitimate.

- (4) His sworn testimony should be unbiased even though it means an adverse decision to the company. (I have given this kind of testimony on more than one occasion - eg. Swan Kauppika vs. Ford Motor Company.) Any company who has the interest of its employees at heart will not ask or expect him to do otherwise.

QUESTION II

Do you believe an industrial organization is justified in refusing or limiting employment to applicants for work who are affected with pneumoconiosis?

A. This depends on several factors.

- (1) The type of pneumoconiosis - obviously if due to inert dust, applicant is employable.
- (2) Past experience as to the number of unjust claims paid and validity of adverse decisions. Granting awards unless reasonable doubt exists or because the company "can afford to pay" besides being dishonest practice, renders a dis-service to both management and employee.
- (3) In cases of simple or uncomplicated pneumoconiosis, most applicants are employable and should be so treated provided some assurance is given that penalty will not be incurred and also provided the employee has taken adequate precautions to safeguard the employee from further harmful exposure to dust.

Discussion

1st direct
MR. KALMYKOW: I would like to ask a question which relates to the problem of whether or not there should be a liability based on the last injurious exposure. Would it be advisable to have prolonged and extensive litigation with the presentation of evidence to the chairman and the determination of the extent of exposure - details which may require considerable time - in order to allocate the cost of liability between each employer in a particular silicosis case? As a practical matter, how would one handle a situation like that?

*Use his
on re-write
with his letter
of 9-29*
MR. HILL: It is my understanding that insurance companies like the system in which the last employer pays all the liability. In Michigan we are able to apportion liability between employers but the entire amount, nevertheless, is charged against the last employer. In order to encourage the employment of individuals who have an abnormal chest roentgenogram we have suggested that there should be joint contributions from the insurance companies. They, however, do not approve that plan.

Although, in Michigan, the last employer is charged with the entire amount of liability, we have not yet had a situation involving a dust exposure of as little as one day. There was a case in which the Ford Motor Company, on employing

Here

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In Michigan our Act provides that the total compensation due is recoverable from the employer who last employed the employee in the employment to the nature of which the disease was due and in which it was contracted. In a contested case proceedings are commenced against the last employer where there was exposure. If liability is established, that employer and his carrier are liable for the payment of the award. If there were prior exposures the last employer can request a hearing and have the other employers brought in for the purpose of apportioning the total liability among the various employers. The apportionment provision as between employers has been in effect since our Act was enacted in 1937 and I don't recall over 5 cases in which it has been necessary to have a hearing to apportion the liability. Generally the prior employers and their carriers get together and fix their proportionate liability by agreement. That is also done in the uncontested cases where it is admitted that the employee is disabled and the last employer or his carrier agrees to pay the compensation due and the prior employers and their carriers agree to contribute a fixed amount to the last employer or his carrier.

A rather unusual situation arose in one case where the Ford Motor Company employing a man told him that the results of his pre-employment X-ray examination would not be available for three days and that if it showed an abnormal chest condition he would be released from employment. The X-ray study did reveal that the man's chest condition was abnormal and therefore he was released. A claim naming the Ford Motor Company and the prior employer where he had had a long exposure to silica dust was then filed. We held the prior employer liable rather than the Ford Motor Company and our Supreme Court said we were correct.

As I understand it, the employers and insurance companies like

Hill

the system which places the full responsibility on the last employer and it certainly makes it more convenient for the employee in proceeding for compensation. We have suggested that in order to facilitate the employment of employees with abnormal chest findings there should be apportionment of liability between the various insurance carriers that have insured a particular employer during the period of the employee's exposure. However, the carriers do not seem to want that provision and prefer that the carrier on the risk at the time of disablement be charged with the entire liability.

OK
Hill

a man, told him that the results of his pre-employment X-ray examination would not be available for three days and that if it showed an abnormal chest condition he would be released from employment. The X-ray study did reveal that the man's chest condition was abnormal and therefore he was released. A claim, naming the Ford Motor Company and another employer, was then filed. The Commission, in its ultimate wisdom, did not use the provision of the last employer but held that the employer previous to the Ford Motor Company was liable. The court agreed with this view.

(use his own re-write with his letter of June 29)

If the last employer is liable for the full amount, he can call in the other employers. That rule has been in effect in Michigan since 1937 and I can recall only four or five cases. Generally the employers settle the claim by agreement, each employer contributing a proportionate amount. Remember that all cases are not contested and that there have been cases in which the employers, while admitting that an individual had a disabling silicosis and that under the law the amount due him should be paid by the last employer, have gotten together and each one has paid a part of the amount. This arrangement is not made under some regulation or formula but the employers arbitrarily fix the amount each one is to pay.

MR. WATERS: Mr. Kuechle, what can be done to minimize unemployability as a disabling factor in dust disease cases?

MR. KUECHLE: That question gives me an opportunity to read a paragraph from a letter from Mr. Harry Nelson, Director of Compensation of the Wisconsin Industrial Commission. I submitted my proposed talk to him because I didn't want to make any errors in speaking about the statute, and in returning the draft to me his concluding comment was this:

"As I read your talk I again wondered why other states flounder around with elaborate and abstruse provisions for partial disability which is really non-existent."

An answer to your question about what can be done to minimize the non-employability of an individual who shows some evidence, clinical or X-ray - it would, of course, have to be X-ray evidence, at least of silicosis - depends entirely upon what sort of law and what sort of administration you have. In Jim Hill's state, Michigan, there is a pretty desperate situation. There is a law and Jim Hill and his associates must administer it the way the legislature put it on the books. Industry in Michigan is, in fact, refusing to employ people who have X-ray evidence of silicosis. Am I correct, Jim?

OK Hill
MR. HILL: Yes. Industry refuses to employ those persons and some foundries discharge them ^{when} ^{X-ray} ^{discloses an abnormal chest condition.} after a periodic examination. That starts the trouble.

OK Kuechle
MR. KUECHLE: ^{Here} ~~Here~~ is a group of employers who, by their actions, say that a man with that amount of silicosis is a hazard to them as far as employment is concerned. The situation is not the Commission's fault and is not the employer's fault. The law is faulty and the administration can do nothing except administer the law the way it is in the books.

In Wisconsin we have no such trouble. In Wisconsin today, in foundries which have dust control measures we know are adequate and which restrict the degree of dustiness to safe limits, industry is employing on the recommendations of Dr. Sander and of other equally qualified physicians individuals who have silicosis, well-recognized nodular silicosis. These individuals, who may come from another state or from another foundry in Wisconsin or from some other industry, are employed because our Commission, as Mr. Nelson pointed out in his letter, takes the position that there is practically no such thing as a disabling silicosis from the standpoint of a man's ability to do his work.

Let us remember why we have compensation laws. Some people seem to be

laboring under the delusion that compensation laws were designed primarily to give people money. That is not correct. I happen to be well acquainted with the committee from the Wisconsin Legislature that drafted the law which was passed in 1911. The chairman of that commission, Mr. Sanborn, was a personal friend of mine and I discussed the matter with him many times. When that bill was submitted to the Legislature, the committee gave several reasons for a compensation law and the number one reason was to reduce the number of accidents and injuries in industrial pursuits. That is the goal we have been trying to follow and that is the goal every administrator should have in mind.

Wealth is created only through the use of human energy in the development of our natural resources; in no other way does one produce real wealth. When, for any reason, a man who is employable is not permitted to work, we are sacrificing part of the wealth, the potential wealth of our nation, and every agency, every individual, ^{that} who plays a part in administering a compensation law should first and foremost look to the one factor of keeping a man at work. It is true that some workers in the foundries have extensive silicosis and can't run a hundred yard dash but how much better it is to let them continue for the rest of their life at their favorite occupation without a wage loss. In Wisconsin, in many of the foundries we insure, we have individuals who had nodular silicosis twenty years ago and are still working. Why not keep them at work rather than give them a few thousand dollars and throw them out of work. That is when they die; that is when they become a burden on society; that is when we must support them through public and private charity. I say that every law ought to be re-examined with the idea of designing it in such a way that in the administration of that law there is every inducement to keep men at work rather than to pay them money.

MR. WATERS: It is not surprising that Mr. Connolly's remarks ran head-on into a subject I discussed at the meeting yesterday afternoon, this germane subject of the question of limitations. I mentioned that in damage actions all states imposed limitations of time within which a suit or claim must be filed and that under most of the compensation statutes there are specific limitations of time when the compensability of the claim must arise or of time within which that claim must be filed. If I understand Mr. Connolly's remarks correctly, he would like to throw some of those limitations statutes out of the window. This is just another area of disagreement that I have with him and with many other members of the panel. I believe it to be of vital interest, however, that Mr. Connolly should have the opportunity to say in a little more concrete form what he thinks. I should like, therefore, to direct to him this question: Should time limitations on the filing of claims for pneumoconiosis be liberalized or abolished?

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Connolly

MR. CONNOLLY: In preparing my paper I had one thing in mind: at no time did I wish to discriminate against people with dust disease. Nowhere in the United States have we a law on our books which states that, for a man who is going to be compensated for the loss of a hand, the time limitation starts from the time of his exposure to what may cause the loss of the hand, yet that is exactly what most of the laws state today on silicosis. They state from the time of last exposure. If you are going to put time limitations on other sections of the act, do the same thing with silicosis, but if you are not going to place time limitations on other impairments, then treat silicosis victims exactly the same way. In other words, I say that if there is going to be that statute of limitations it should be from the time of disablement, exactly the same as it is for the loss of a hand - from the time a man loses a hand he would have

two years to report that accident.

There will always be disputes if you state the time of last exposure because the man may go on for two or three years without knowing of his impairment or he may be careless about it and then, when he is disabled, come in and make his claim only to have it disallowed on the basis of the statute of limitations. When I administered the law in Rhode Island, I had that problem and, strange as it may seem, I made the administrative ruling that the time limitation meant from the time of disablement, yet not a single case was challenged on the basis of that ruling. Every claim was paid, and we ^{had} quite a few - I believe there were about 70 - insurance companies writing insurance in Rhode Island. I just want the person with silicosis to be treated exactly the same way as an individual with any accident or with any dust disease. I am using the word silicosis when perhaps I should be using the term pneumoconiosis, and if that condition can't be treated the same as any other accident, then I say abolish the state of limitations.

MR. KUECHLE: I do not consider Mr. Connolly's comparison an appropriate one. In one case you are dealing with a progressive disease; in the other case you refer to a man whose hand has been chopped off. I agree with Mr. Connolly, however, that if you are going to have a statute of limitations at all, it ought to be a very liberal statute. If you want to keep these men at work, they must have some reasonable assurance while they're working that if eventually they break down they are going to be taken care of.

MR. CONNOLLY: I agree with that.

MR. KUECHLE: In Wisconsin we have attempted to handle the situation in such a way that a man has practically no statute of limitations ever applying against him.

MR. BARDICH: Dr. Hardy told us, earlier in this symposium, that cases of beryllium disease were turning up ten years after the termination of exposure. From my past experience I anticipate that two years from now she will be telling us that cases are turning up twelve years after the last exposure. How then is one going to set any kind of a definite time limit?

DR. RICHARDS: One of the greatest difficulties we are encountering on this panel today is the fact that we have written too many laws in ignorance. My good friend, Otto Wiesley, brought before you some points on this subject. Our state, Utah, is just a little state but we had twenty years of thorough investigation before we wrote an occupational disease law. When we were ready to write that law we brought in labor, management, attorneys, environmental engineers - they even permitted a couple of us doctors to creep into the crowd. With that background as a basis we all sat down with equal responsibility and wrote a law. You may not agree with it but it has worked. If you don't agree with it, then give us some reason why it isn't a reasonable law.

MR. CONNOLLY: I didn't disagree with it; I said it is almost impossible.

DR. RICHARDS: It isn't impossible because it has been done. With that as a background, when we confront the law we have today, labor is just as responsible for that law as is management or the attorney or the health engineer or the doctor. So we have on our statutes a law that, when submitted to our legislature, was passed without a single dissenting vote, and if labor now wishes to condemn this law we say: "All right, gentlemen, you are condemning the law that you helped to build after twenty years of investigation, after a thorough analysis of all your health problems as accurately as we could make it, after a health investigation throughout our state during which thousands of films were taken." So it is

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OK

OK
Richards

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Connolly

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OK
Richards

evident that our pneumoconiosis problem was not met indiscreetly and without some foresight. I feel, therefore, that if we could establish a pattern whereby all the parties involved sit down in council and discuss what they want to do, a lot of these problems would be worked out.

OK
Richards
Many of the problems that have come before this group today will only be worked out educationally. The success of such an operation will be measured by how well the boards that are to administer these laws can get along with management and with labor and can deal with all the other factors involved. If we can sit down, in the united capacity of a cooperative and amalgamated group, and study our problems, there will be an agreeable solution to all these things.

OK
Connolly
MR. CONNOLLY: One of the greatest problems in the United States today is, as Dr. Richards very well put it, poor laws. We in the Bureau of Labor Standards have been trying to develop a so-called model law that will bring at least some of our states in line with other states. Perhaps the governor of Dr. Richards' state has control of the legislature but when I went in as Director of Labor in Rhode Island we had a very bad law and as the Director of Labor I submitted 23 amendments to correct some of the abuses in that law. I succeeded only in making that law more hodge-podge than it was in the beginning. But we did correct some things here and there.

I then went to the governor of the state and asked him to set up a commission to study the law and to bring back to the legislature a report on what a model law should contain. The report was made and the bill has been before the legislature for three long years. We still have the hodge-podge law in Rhode Island.

We can go straight across this country yet we will not find two workmen's compensation laws that are anywhere near alike. One of the greatest fights I'm having right now in Washington is to prevent the federalization of workmen's compensation. That attempt is not coming from the federal government; it is

coming from people outside the government. I have taken the stand that workmen's compensation cannot be federalized and for that reason I oppose federalization. The International Association of Accident Boards and Commissions, which is made up of all the workmen's compensation commissioners in this country - I am the secretary-treasurer of that organization - have asked that a meeting of all workmen's compensation commissioners be called in Washington for the purpose of setting up this model law. But even there we can't get agreement. Some people who want to come into that meeting have views so far apart that we would succeed only in having another knock-down, drag-out fight.

We are all working toward the end of simplifying workmen's compensation laws and when I said it was impossible I meant that there are too many people in our state legislatures who differ in their views on what should be included in the law and what should be omitted. That is the main difficulty and that is why I said it was impossible. If we could control the legislatures we certainly could get up a good model law. The present law in Mississippi is one of the best laws in that part of the country as it was just enacted in 1948. That law was drawn up in the Bureau of Labor Standards. In Mississippi changes in the law were made - changes we didn't like - but it came out in pretty good form. Mississippi was the last state in the union to enact, in 1948, a workmen's compensation law. That law represents our closest approach to putting into one act all the things we believe would be good for all parts of the nation.

MR. BARNAKO: In your opposition to the statute of limitations are you talking about exposure or about disability? Are you opposed to limitations in general or are you considering only the problem of whether the time limitation should extend from the last exposure or from the date of disability?

Not
seen
Hearings
necessary

OK
● Connolly
MR. CONNOLLY: My position is that the limitation should extend from the date of disability.

MR. HILL: The workmen's compensation laws are designed to provide benefits for disability or death that result from an injury which arises out of and in the course of employment. The term injury includes diseases and under an all-inclusive type of occupational disease coverage it would include any kind of disease that would result from exposure to beryllium, ^{it would be included} and if beryllium was listed in the schedule ^{under so-called schedule coverage.} ~~it also would be covered.~~

Somebody will have to take care of these people who become disabled and die from berylliosis or who suffer any other disabling result from exposure to beryllium. Either they must be paid benefits under the compensation law or ~~the state or federal government~~ ^{the state or federal} they must be paid benefits by ~~somebody else~~ ^{be paid} ~~under some plan.~~ ^{government or somebody else.} I submit that they should ~~get them under~~ ^{be paid} workmen's compensation benefits ^{benefits} and that the only possible limitation you can have would be a limitation from the date of disablement. If it can be established that the delayed reaction is due to the employment, I agree that there should be some limitation, from the date of disablement, in which a claim should be made or proceedings started. But certainly I do not believe that a worker's claim should be barred merely by the lapse of time. Even if the effects of beryllium are not felt or do not cause disability until 10 or 12 or 15 years after exposure, nevertheless, if the individual's condition arises out of and in the course of his employment, he should be compensated.

MR. WATERS: This is an important question, one upon which I personally have strong views. Mr. Kuechle and Dr. Hamlin, in your opinion should employees be permitted to sign waivers when continued exposure would definitely jeopardize

their future health? Under the statute of many states provision is made for an employee to sign a waiver which would limit or eliminate the continuing monetary liability of the employer under the statute.

OK
Kuechle

MR. KUECHLE: Personally, I believe that waivers are an abomination. We have never permitted a policy holder of ours knowingly to take a waiver from an employee. If a man's condition is such that he should not receive further exposure, the only humane thing to do is to remove him from that employment. Most employees do not understand and never will understand the implications of the signing of a waiver. I believe it's ~~little~~ short of being criminal to permit the taking of waivers.

OK
Corrected
by
Hamlin
on July 15, 53

DR. HAMLIN: Most of us would agree with Mr. Kuechle's view that it isn't fair to ask any employee to sign a waiver ^{concerning} of anything that might affect his future ^{influence} health. There are other things, however, that might ~~affect~~ the situation. If an employee could be put to work with the assurance that he would not suffer any further exposure - I mean through the use of industrial hygiene and dust control measures and the ~~elimination~~ ^{dusts} of all hazardous exposures - then, of course, a waiver wouldn't be necessary. And I see no objection in allowing an employee to continue at a job where there might be some ^{risk} ~~hazard~~, provided the exposure is one that is well controlled.

This ~~point~~ brings up the question of maintenance of dust control measures. We know perfectly well that dust concentrations can be reduced to a ~~satisfactory~~ safe level but if that condition is not maintained adequately over a long period of time and constantly kept in mind, there is no guarantee that the employee will not have an exposure in the future.

Legally, as far as I understand it, the signing of a waiver is not permissible and I do not believe that it should even be considered.

MR. WATERS: Dr. Orenstein, what is the practice in South Africa as to the use of waivers?

DR. ORENSTEIN: The signing of such a waiver is forbidden by statute. The underlying philosophy is that while you may prejudice your own rights you have no right whatever to prejudice the rights of your dependents.

MR. WATERS: I wish to direct a question to Commissioner Wiesley and to Commissioner Hill: From your experience what is the most difficult problem in determining contested pneumoconiosis cases?

MR. WIESLEY: For the record my answer is, I don't have any. The first seven years of my experience were just a hit-and-miss proposition and I couldn't get Dr. Richards to help me take or read the X-rays and arrive at a diagnosis. We just had to accept the report sent in from some unreliable source in which the diagnosis was based entirely on employment history with no knowledge of whether the man did or did not have exposure. Since we have had our medical panel we have proven rather conclusively that we have a good many doctors in Utah who don't know silicosis from measles. You may be interested to know that twenty men who have been processed by our panel of experts have died and, because of our compulsory autopsy law, an autopsy was done on every one of them. We were not satisfied with only the panel diagnosis. The records of many of the cases were sent to Canada, to Dr. Sander, to Dr. Pendergrass, and in every instance they verified the diagnosis of our panel and, of course, the autopsy findings verified the diagnosis of both groups. I say, therefore, that at the present time we don't have any difficulty.

Regarding the question of exposure we do not have any problem. In 1941, in the very first case I processed, I personally heard it and wrote the decision.

The case went to the Supreme Court and my decision was affirmed. The medical testimony was quite unsatisfactory - all we had was the radiologist's reading of the film and his diagnosis of silicosis and inactive tuberculosis. The State Insurance Fund didn't do anything about the matter so the record stood. The superintendent of the mine testified that there was some silica rock in the mine and the evidence showed that the man had worked only in that place. I said, on the record this man has made a prima facie case. He has the disease, he is totally disabled, there is the only place where he worked, the evidence shows that there was exposure to free silica dust. Therefore we made the award. The Supreme Court agreed with our decision.

All our cases, so far, have come from mines. Our Supreme Court takes judicial notice of the fact that in every non-ferrous mine and every coal mine in Utah the miner is exposed to free silica dust. Therefore we don't have any problem on this point.

We have that 30-day provision, and the last employer in whose employ the individual was exposed to free silica dust is responsible. He never takes the trouble any more to make dust counts. We don't have to go into the evidence; we don't have any problem.

OK
(correction)
by Hill
MR. HILL: I wish I could agree with Mr. Wiesley. Our biggest problem is simply this: Is the man disabled? I can illustrate the problem best by a couple of typical situations.

Here is a man, in his fifth decade, who has been working in a foundry for 20 or 30 years. Suddenly he says, "I can't take it any more, I'm tired out, I'm short of breath walking upstairs. My chest feels tight and I cough all the time". So he just quits. He hasn't ^{been} ~~seen~~ to a lawyer ^{yet but} ~~and he doesn't consult one.~~ But

he is going to ~~one~~ pretty soon.

Then he files a claim. He simply says he can't work and, of course, we're in this disputed medical field again and ask, is the man disabled? There may be some disagreement about the X-ray diagnosis but frequently the doctors will say that the individual has early silicosis with some fine nodulation but that the condition does not interfere with his ability to work. Nevertheless the man says he can't work and the physician who testifies for him says it would not be advisable for him to return to work ^{in the foundry.}

Conclusion

67 Hill
We have also the situation of a man, probably in the same age group, who has been working a long time and who gets a stubborn cold - maybe he has bronchitis, maybe pleurisy. The family physician treats him during the winter and in the course of the treatment sends him to a radiologist. The report by the radiologist states that the man has early silicosis with perhaps a minimal amount of fibrosis or possibly ~~with~~ more than a minimal amount. The family physician then informs the man, "You have dust in your lungs, you shouldn't go back to work at the same job. I'll write a note and you take it to your employer when you return to work". The man takes the note to ~~the~~ employer, who then has the man examined by the plant physician and X-ray pictures taken. After receiving the report of the studies the employer tells the man, "You have minimal silicosis but it won't interfere with your returning to the same job. Either you work at that job or you have no job at all."

How in the world does one decide those cases? They involve almost insurmountable problems. In the first case illustrated there is a man with a good work record of many years who suddenly quits. Are we going to say that he is not disabled because the X-ray evidence doesn't fit the pattern of the man who is supposed to be disabled from silicosis? And in the second case are we going

to assert that the man doesn't have the right to rely upon the advice of his family physician?

MR. WATERS: It is a good thing that the question was not directed to a lawyer - the question as to what one considers to be the most difficult problem dealing with contested pneumoconiosis cases - because the reply might not reflect too much credit upon some of the Commissioners.

Many of the questions that have been presented are highly controversial and I'm certain that there still exists differences of opinion among us with respect to them. I think it is only fair to say that we are indebted to The Saranac Laboratory for this opportunity to receive such authoritative information and advice about these problems with which all of us must deal.

Brake Shoe

AMERICAN BRAKE SHOE COMPANY
MEDICAL DEPARTMENT
2501 BLUE ISLAND AVENUE
CHICAGO 8, ILLINOIS

September 9, 1952

Arthur J. Vorwald, M. D., Director
The Trudeau Foundation and
The Saranac Laboratory
Saranac Lake, New York

Dear Art:

I have revised my talk along the lines you suggested and also since writing you I have added another case record because it points out very definitely the type of thing I am talking about. Will you kindly therefore insert the attached three pages, numbers 3, 4, and 5 to replace page 3 of the manuscript you already have. Kindly number the succeeding pages to conform with these.

I have also attached my replies to the questions I submitted in the event you wish to see these before the panel discussion.

Yours sincerely,

L. E. Hamlin

L. E. Hamlin, M. D.
Medical Director

leh/em
Encls.

cc: Mr. Theodore C. Waters, Baltimore, Md.

August 28, 1952

L. E. Hamlin, M.D.
Medical Director
American Brake Shoe Company
Medical Department
2501 Blue Island Avenue
Chicago 8, Illinois

Dear Doctor Hamlin:

In Doctor Vorwald's absence while on a business trip, I wish to acknowledge and thank you for your letter of August 26 enclosing a copy of your talk for the panel discussion of our Seventh Saranac Symposium. This is the first Symposium paper we have received and I know that Doctor Vorwald will be very happy to have it on his return to the laboratory the first of the week.

Sincerely yours,

Lillian R. Elinn (Mrs. A.B.)
Executive Secretary to -
Arthur J. Vorwald, M.D.
Director

LRB:inf

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Director

LRB:iaf

Brake Shoe

AMERICAN BRAKE SHOE COMPANY
MEDICAL DEPARTMENT
2501 BLUE ISLAND AVENUE
CHICAGO 8, ILLINOIS

August 26, 1952

Arthur J. Vorwald, M. D.
Director, The Trudeau Foundation
and The Saranac Laboratory
P. O. Box 551
Saranac Lake, New York

Dear Art:

I am enclosing herewith a copy of my talk for the panel discussion, as you requested. I hope it meets with your approval. I am also sending a copy to Ted Waters.

I plan to show about a dozen slides to demonstrate the variety of conditions that may simulate silicosis.

Kindest regards.

Yours sincerely,

Ham

L. E. Hamlin, M. D.
Medical Director

leh/em
Encl.

BY MR. WATERS:

I am pleased to advise the audience that the airplane schedule has been changed. The weather apparently has cleared and there is not the quite pressure of time that we were subject to when we started the meeting.

It is always the chairman - the prerogative the the chairman of any meeting to reverse his field, and I'm going to reverse my field today. There have been a number of prepared questions to be directed to the panel, but I think the audience comes first and we will welcome any questions that you may wish to direct to any individual member of the panel. In propounding those questions, kindly give your name, the company with which you are affiliated or your address and the participant of the panel to whom you wish your question directed. If you run short of questions, I've got one or two that I want to slap to the members of the panel myself, but at the moment, I invite

your questions. Any questions, gentlemen?

BY MR. KALMYKOW:

I'd like to address this question to the last speaker, which relates to the question of whether there should be a liability based on last injurious exposure or not, and Mr. Keuchle, if I'm not mistaken, didn't express an opinion on that subject himself. I was wondering if the last speaker would think it advisable to have a long and extensive litigation and evidence having to be presented to the chairman, the extent of exposure under all employers which may spend a considerable period of time, to be able to allocate cost of liability between each in a particular silicosis case, just as a practical matter, how would you handle a situation like that?

BY DOCTOR HAMLIN:

That's a pretty long, involved question for me, and I don't claim, as I said, to know much about the administration. I think that's a question that might be directed better to one of the other speakers. I'm not sure that I quite understand just what you were - what the gist of your question is.

BY MR. WATERS:

Commissioner Hill, do you mind if Commissioner Hill answers that?

BY MR. KALMYKOW: No.

BY COMMISSIONER HILL:

I may not be able to answer it, but I got the gist of it. You say does he oppose a system whereby the last exposure pays all the liability? Now, as I understand it, the insurance companies like that system. In Michigan, we have - we're able to apportion liability between employers, but the last employer is stuck, but we've suggested that in order to facilitate employment of employees with abnormal chest X-rays, that there should be contribution between insurance companies, but they don't want that.

Now, in Michigan, the last employer is stuck with the entire amount. We have not had any situations that involve as little as one day. The Ford Motor Company, in one case, employed a man and told him on pre-employment that they couldn't get the result of his X-ray examination for three days, but if it showed that he had abnormal - an abnormal chest condition, that he would be released and so as it did, he was released. Then a claim was filed naming the Ford Motor Company and another employer, and the commission in their ultimate wisdom, didn't use the provision of the last employer, but held the employer previous to the Ford Motor Company, and the court said we were correct.

But your whole - the whole problem here is that the - if the last employer is stuck for the full amount, he can call in the other employers. Now, that rule has been in

effect in Michigan since 1937 and I can recall only three to five cases, generally the employers settle it by agreement, and say you contribute so much, you contribute so much, you contribute so much.

Now, remember that all cases are not contested and there have been cases in which employers have contributed where they have agreed that the man had a disabling silicosis and the amount payable should be paid by the last one under the law, but the boys getting together, the several employers, and say, I'll pay this much and I'll pay that much. They do it not under some regulation, I mean they just arbitrarily fix the amount each is going to pay out of the total amount due. There is no particular formula. Now, I don't know if that answers your question or not.

BY MR. WATERS:

Thank you, Jim. I am going to again exercise another prerogative and direct a question to one of the members of the panel that has been the subject of discussion in the course of the meeting yesterday afternoon and this morning. Ben Keuchle happens to be one of my father confessors in this field. This question is not prepared for him, but I'd like to get his reaction, both from the standpoint of management and an insurance carrier, with respect to this question.

Ben, what can be done to minimize the unemployment

as a disabling factor in dust disease cases? Would you say a word about that?

BY MR. KEUCHLE:

That gives me an opportunity to read a paragraph from a letter from Mr. Harry Nelson, the Director of Compensation of the Wisconsin Industrial Commission, which I neglected to read when I was up here before. I submitted my talk to him, because I didn't want to make any errors in speaking about the statute, various statutes of limitations that we have in our law, and his concluding paragraph, in sending the draft back to me is this:

"As I read your talk, I again wondered why other states flounder around with elaborate and obstruse provisions for partial disability, which is really not existent."

Now, your question, what can be done to minimize the non-employability of an individual who has some evidence, clinical or X-ray, naturally would have to be X-ray, at least of silicosis - that depends entirely on what sort of a law you have and what sort of administration you have.

Now, I know over in Jim Hill's state, the great State of Michigan, you have a pretty desperate situation. They have a law, and Jim Hill and his associates have got to administer it the way the legislature put it on the books. Industry is, in fact, refusing to employ people in the State of Michigan who have X-ray evidence of fibrosis, am I right, Jim?

BY COMMISSIONER HILL:

Yes, I mean, they refuse to employ them, some of the foundries discharge them, just say, you're out of a job on a periodic X-ray examination, that starts the trouble.

BY MR. KEUCHLE:

Now, here is an employer group who, in fact, by their actions, say that a man with that amount of silicosis is a hazard to them as far as employment is concerned. It isn't the Commission's fault, it isn't the employer's fault, the law is faulty, and the administration can't do anything but administer the law the way they have it on the books.

Now, we have no such trouble in Wisconsin. You gentlemen and ladies here would be amazed, and Doctor Sander would verify this if he's here, when I tell you that in Wisconsin today, in our foundries where we know dust control is adequate, we're keeping well within safe limits, industry is employing on the recommendations of Doctor Sander and other equally qualified physicians, men who have silicosis today, who may come from another state or from another foundry in the State of Wisconsin or from some other industry that we will recognize - that have well recognized nodular silicosis, because our Commission, as Mr. Nelson pointed out in this letter, takes the position that there is practically no such thing as a disabling silicosis from the standpoint of the man continuing to do his work. Now, let's remember

what this -- am I talking too long?

BY MR. WATERS:

Go ahead.

BY MR. KEUCHLE:

Let's remember what we have compensation laws for. Some people seem to be laboring under the delusion that compensation laws were primarily designed to give people money. That is not correct. I happen to be well acquainted with the committee from the Wisconsin Legislature, in 1909, that drafted the law which was passed in 1911. The Chairman of that Commission, Mr. Sanborn, was a personal friend of mine. I talked with him many times, and when that bill was submitted to the legislature, they gave - the Committee gave several reasons for a compensation law, and number one reason was to reduce the number of accidents and injuries in industrial pursuits.

Now, that is the goal we have been trying to follow, and that is the goal every administrator should have in mind. Wealth is only created through the use of human energy in the development of our natural resources, and in no other way do you produce real wealth. When a man is not permitted to work for any reason, who is employable, you are sacrificing part of the wealth, potential wealth of this nation of ours, and every agency, every individual who plays a part in administering a compensation law, should,

first and foremost, look to the one factor of keeping a man at work.

Now, it's true that some of these fellows in the foundries, have extensive silicosis and they can't run a hundred yard dash, but how much better to let them go on for the rest of their life at their favorite occupation without a wage loss, and we've got people in Wisconsin in a lot of the foundries we insured, who had nodular silicosis twenty years ago and they are still working. Why not keep them at work rather than to give them a few thousand dollars and throw them out of work? That's when they die; that's when they become a burden on society; that's when we've got to support them through public and private charity. I say that every law ought to be looked at again with the idea of designing it in such a way that in the administration of that law, there is every inducement to keep men at work rather than to pay them money.

BY MR. WATERS:

Thank you, Ben. It's not surprising that Bill Connolly's remarks ran head-on into one of the subjects that I happened to discuss at the meeting yesterday afternoon. Perhaps Bill wasn't here, but it dealt with this other germane subject of the question of limitations. In summary, you recall that I mentioned that in damage actions, all states imposed limitations of time within which suit or

claims must be filed and under most of the compensation statutes, there are specific limitations of time when the compensability of the claim must arise or the time within which that claim must be filed.

Now, if I understood Bill correctly in his remarks, he'd like to throw some of those limitations statutes out of the window. This is just another area of the many disagreements that I have with him, and many of the other members of the panel, but in the light of that discussion, and I think it is of vital interest to the record of this symposium, that Bill should have the opportunity to say a little more concrete form, what he thinks. To that end, I'd like to direct to him this question:

Should time limitations on the filing of claims for pneumoconioses be liberalized or abolished? Will you say a word about that, please, Bill?

BY MR. CONNOLLY:

Glad to, Ted. In answering that question, and when my paper was prepared I had one thing in mind, that at no time did I want to discriminate against people with dust diseases. We haven't, anywhere in the United States, a law on our books, which says that a man who is going to be compensated for the loss of a hand, the time limitation starts from the time of his exposure to what may cause the loss of the hand, and that's exactly what most of the laws

say today on silicosis.

They say from the time of last exposure. Now, if you're going to put time limitations on other sections of the act, then do the same thing with silicosis, but if you're not, then treat silicosis victims exactly the same way, in other words, I would say that if there is going to be that statute of limitations, then it should be from the time of this disablement, exactly the same as if he lost his hand, from the time that the hand comes off, he would have two years to report that accident.

Now, there is always going to be disputes if you say the time of last exposure, because the man may go two or three years without knowing it or he may know it and be careful about it, or be careless about it and then, when he is disabled, come in and make his claim, and it's disallowed on the basis of the statute of limitations.

Now, I had that problem when I administered the law in Rhode Island, and strange as it may seem, I made the administrative ruling that that meant from the time of the disablement and not one case was challenged on the basis of that. Every claim was paid and we have quite a few insurance companies writing insurance in Rhode Island. I think were some 70, and so, that's my position on it. I just want the silicosis person treated exactly the same way as any other accident or any dust diseases.

I'm using the word silicosis perhaps when I should be using that pneumoconiosis and if they can't be treated the same as any other accident, then I say abolish the statute of limitations.

BY MR. WATERS:

Very well done.

BY MR. KEUCHLE:

Now, can I fight?

BY MR. WATERS:

Sure, come ahead, three minutes to fight.

BY MR. KEUCHLE:

I don't think Bill's comparison is appropriate at all. In one case, you're dealing with a disease that is progressive; in another case you're having a man's hand chopped off. However, I do agree with Bill that there ought to be, if you're going to have any statute of limitations at all, a very liberal statute. It ties right in with what I said a moment ago, if you want to keep these men at work, they've got to have some reasonable assurance while they're working that if eventually they break down they're going to be taken care of.

BY MR. CONNOLLY:

Well, I agree with that.

BY MR. KEUCHLE:

We have attempted, in Wisconsin, to handle that

situation in such a way so that a man practically has no statute of limitations ever applying against him. Now, some of you may want to write my president and tell him I ought to be fired.

BY MR. BARDICH:

Mr. Waters, may I ask a question?

BY MR. WATERS:

You may, Mr. Bardich.

BY MR. BARDICH:

We were told on Monday that cases of beryllium disease were turning up ten years after the termination of the exposure. From my past experience, I'll bet a cookie that two years from now, she'll be telling us that cases are turning up twelve years from the last exposure. Now, on that basis, how are you going to set any kind of a definite time limit?

BY MR. WATERS:

Since I am not a member of the panel, I'm going to ask one of my good brethren to answer Mr. Bardich's question. Jim, I see Doctor Richards seems - would like to comment upon it, is that correct, Paul?

BY DOCTOR RICHARDS:

Yes. I would say one of the greatest difficulties that we are encountering on this panel today is that we have written too many laws in ignorance. Now, to come back with

my good friend, Otto Wisely, and recapitulate somewhat of a pattern of the thing that he brought before you, now, we are just a little sagebrush state out there, folks, but we have twenty years of thorough investigation before we wrote an occupational disease law. When we got ready to write that law, we brought in labor, we brought in management, we brought in the attorneys, we brought in the environmental engineers and lo and behold, they permitted a couple of us doctors to creep into the crowd.

Well, now, then with that background, that as a basis, we all sat down with equal responsibility and wrote a law. Well, you may not agree with it, but the damned thing has worked. Now, then if you don't agree with it, then give us some reason why it isn't reasonable.

BY MR. CONNOLLY:

Well, I didn't disagree; I said it's almost impossible.

BY DOCTOR RICHARDS:

It isn't impossible, because it's been done. Now, then with that as a background, when we confront the law that we've got today, labor is just as responsible for that law as management or as the attorneys or as the health engineers or as the doctor. So we've got a law on our statutes that when it went before our legislature, there was not one single dissenting vote in that legislature, and now then

if labor wants to come and condemn this law, we say, all right, gentlemen, you are condemning the law that you helped to build after twenty years of investigation, after a thorough, after a thorough analysis of all of our health problems, as accurately as we could make them, in a health - in a health investigation throughout our state, which resulted in the taking of thousands of films, so our pneumoconioses problem was not met indiscreetly and without a little foresight. Therefore, I feel that if we could establish a pattern whereby all of the parties involved sit down in council on what they want to do, I believe a lot of these problems would be worked out.

Now, I think a lot of the problems that have come before this group today will only be worked out educationally. The success of the operation of this thing will be how well can our boards that are to administer these laws get along with management, with labor and all the other factors involved. If we can still sit down, in the united capacity of a cooperative and amalgamated group in studying our problems, there is still an agreeable solution to all of these things.

BY MR. WATERS:

Gentlemen, you know, I wish that this plane that's supposed to leave Saranac, would leave at five o'clock in the morning instead of five o'clock in the afternoon. I

predicted today we were going to have a slugging match, and Bill Connolly just whispered in my ear that he couldn't take everything that Doctor Richards had said and he wants to reply. I am going to let him reply, but I'm sorry to tell you that when he is through, I must again exercise my prerogative and make a few remarks. Time is running out and I would like to hear from Bill, for the record.

BY MR. CONNOLLY:

One of the greatest problems we have in the United States today is, as Doctor Richards very well put it, poor laws. We, in the Bureau of Labor Standards, have been trying to develop a so-called model law that will at least get some of our states in line with others. Now, if you take perhaps the Governor there has control of his legislature, but let me tell you that when I went in as Director of Labor in Rhode Island, we had a very bad law, and as the Director of Labor, I submitted twenty-three amendments to correct some of the abuses in that law. I just succeeded in making that law more hodge-podge than it was in the beginning.

Sure, we did correct some things here and there. I then went to the Governor of the State and asked him to set up a commission to study the law and to bring back to the legislature a report on what a model law should contain. He did that. That bill has been before our legis-

lature for three long years. We still have the hodge-podge law in Rhode Island.

Now, we can go straight across this country and we can't find two workmen's comp laws that are anywhere near alike, and one of the greatest fights I'm having right now, if you please, in Washington, is to prevent the federalization of workmen's compensation.

Now, that is not coming from the federal government. It is coming from people outside the government, and I have taken my stand that the workmen's compensation can not be federalized, and for that reason, I oppose it. The International Association of Accident Boards and Commissions which is made up of all of the workmen's compensation commissioners in this country, and I am the Secretary-Treasurer of that organization, they also have asked to call a meeting in Washington, of all workmen's compensation commissioners for the purpose of getting up this model law. We can't even get agreement there. We have some people that want to come into that meeting that - and their views are so wide apart and so far apart that we'd only succeed in having another knock-down, drag-out fight where we couldn't do the job.

Now, we're all working toward the end of simplifying workmen's compensation laws, but when I said it was impossible, I say that there are too many people in our state legislatures who differ in their views on what should be

here or what should be there in a law, and that is the main difficulty, and that is why I said it was impossible. If we could control these legislatures, fine, we certainly could get up a good model law, the law that is now in Mississippi is one of the best laws around that part of the country, I guess, of any of the laws, for they just enacted their law in 1948. That law was drawn up in the Bureau of Labor Standards. When it got down there, they did make changes in it, changes that we didn't like. However, it came out in pretty good form and they were the last state in the union to enact a workmen's compensation law, and as I say, it was in 1948, but that's the closest we've come to being able to put into one act, all of the things that we think are good throughout the nation.

BY MR. WATERS:

Gentlemen, word has come to me that I may have been mistaken, after all, in honest confession is good for the soul, and I must ask my friend, Doctor Richards, with respect to this, the time for the departure of the plane. The reason I ask that is that word has come to me that a number of those in the audience would like to stay on for further discussion. Paul, do you know about the plane?

BY DOCTOR HUSSEY:

The bus leaves the hotel at 5:35 daylight time.

BY DOCTOR VORWALD:

That's the time on the clock there.

BY DOCTOR RICHARDS:

We have one hour before the bus leaves.

BY MR. WATERS:

Well, then I think I was perhaps a little premature in my remarks. I understood it was five o'clock. Therefore, with your forbearance, let's continue this discussion a few moments. Are there any other questions you would like to direct to the panel?

BY MR. BARNAKO:

Mr. Connolly, in your opposition to the statute of limitations, are you talking about exposure or disability?

BY MR. WATERS:

Do you mind addressing the question so the audience can hear? This is very important and I'm sure that this discussion will be helpful.

BY MR. BARNAKO:

My only question is your opposition to the statute of limitations, is whether you're opposed to them generally or whether it's a problem of from the last exposure or date of disability?

BY MR. CONNOLLY:

Date of disability, that's my position.

BY MR. BARNAKO:

And that's what you would desire?

BY MR. CONNOLLY:

That's right.

BY MR. WATERS:

Are there any more questions from the floor?

Commissioner Hill wants to say something; all of them want to talk.

BY COMMISSIONER HILL:

Well, I'll tell you, the reason that he started to ask me to try to attempt to answer the gentleman's question about disability, from delayed disability from exposure to beryllium, and that was called to my attention and that is why I asked that he return. Now, somebody is going to have to take care of these people that become disabled and die from berylliosis or any other disabling result from exposure to beryllium.

The workmen's compensation laws are designed to provide benefits for disability or death that result from an injury which arises out of and in the course of the employment and the term injury, includes diseases and under an all-inclusive type occupational disease coverage, it would include any kind of a disease that would result from exposure to beryllium and if beryllium was listed in the schedule, it would also be covered.

Now, you're either going to have to pay them compensation benefits under the compensation law or the state

or the federal government or somebody else is going to have to pay benefits under some plan.

Now, I submit that they should get it under workmen's compensation, and that the only possible limitation you can have would be a limitation from the date of disablement. Now, the delayed reaction, nevertheless, if you can establish that it's due to the employment, that's the test. If it can be established that it's due to the employment, then there, I agree that there should be some limitation from the date of disablement in which claim should be made or in which proceedings should be started, but certainly I don't think that a worker should be barred merely by the lapse of time. If the effects of beryllium are not felt or do not become disabling until ten or twelve or fifteen years later, nevertheless, if the condition arises out of and in the course of the employment, then it should be compensated.

BY MR. WATERS:

Here is an important question, gentlemen, one with which I, upon which I personally have had a strong opinion. I'm going to direct it to two members of the panel, Ben Keuchle and Roy Hamlin. In your opinion, should employees be permitted to sign waivers when continued exposure to - would definitely jeopardize their future health; under the statute of many states, provision is made for an employee

to sign a waiver which would limit or eliminate the continuing monetary liability of the employer under the statute. May I repeat the question: Should employees be permitted to sign waivers when continued exposure would definitely jeopardize their future health? Will you say a word about that, Ben?

BY MR. KEUCHLE:

Personally, I think waivers are an abomination. We have never permitted a policyholder of ours, knowingly, to take a waiver from an employee. If a man's condition is such that he shouldn't remain at further exposure, there is only one humane thing to do and that's -- what happened -- there is only one humane thing to do and that is take him out of the employment, but to take away, which most employees don't understand when they sign what they call a waiver, but the implications of it they do not understand and never will understand, and I think it's nothing short of criminal to permit the taking of waivers.

BY MR. WATERS:

Doctor Hamlin, will you comment for just a moment upon that subject?

BY DOCTOR HAMLIN:

I think that most of us would agree that, with what Ben just said, that it isn't the fair thing to ask any employee to sign a waiver, anything that might possibly

prejudice his future health. There are other things, however, that might effect that.

If an employee could be put to work with the assurance that he would not suffer any more exposure and I mean through the use of industrial hygiene and dust ventilation and cutting down all hazardous exposures, then, of course, a waiver wouldn't be necessary. I see no object, whatsoever, and I don't think that you can - you can take a chance even where you have exposure that's pretty well controlled, in allowing an employee to continue where there might be some hazard.

Now, that brings up the question of maintenance on that job. We know perfectly well that you can reduce dust concentrations to a satisfactory safe limit and can carry on, but unless that is maintained adequately over a long period of time, and constantly kept in mind, you can not guarantee that that employee is not going to have some future exposure. Legally, as far as I understand, it's not permissible and I don't think that it should even be considered.

BY MR. WATERS:

Glad to say that these gentlemen agree with me. Doctor Orenstein of South Africa is with us, and if he'd be kind enough, I'd appreciate it if he'd say just a word as to the practice in his country, as to the use of waivers,

Doctor, would you be good enough to do that? Do you mind coming to the platform, please, sir?

BY DOCTOR ORENSTEIN:

It is, by statute, forbidden to sign such a waiver. The underlying philosophy that you may - that you may prejudice your own rights, but you have no right whatever to prejudice the rights of your dependents.

BY MR. WATERS:

Thank you, Doctor. I'm going to pose one more question to the two commissioners who are present. I think it's an important question, because of the symposium here, and Now, general interest and concern with those concerned with compensation laws.

The question is this. I direct it first to Commissioner Wisely and, secondly, to Commissioner Hill, and if Bill Connolly wants to get mad with them, I'll listen to him for a half a minutes. Commissioners, from your experience, what is the most difficult problem in determining contested pneumoconioses cases? Otto, would you say a word about that?

BY COMMISSIONER WISELY:

Let me put this in the record: My answer is, Hell, I don't have any. You asked me to speak from experience. Frankly, the first seven years of my experience have been just a hit and miss proposition, and I couldn't get

Doctor Richards to help me make or read the X-rays and arrive at a diagnosis, we just had to accept the report of some silly ass sent in, basing his diagnosis entirely on employment history, no knowledge of whether the man did or did not have exposure, and since we have had our medical panel, we have proven rather conclusively that we have a Hell of a lot of doctors in Utah who don't know silicosis from measles, and you might be interested to know, that because of our autopsy, compulsory autopsy law, twenty men who have been processed by our panel of experts died. We did a post on every one of them. We weren't satisfied with just our panel diagnosis. I sent many of them to Canada, some to Doctor Sander, some to Doctor Pendergrass, and in every single instance, they verified the diagnosis of our panel and, of course, the autopsy verified the diagnosis of both groups, so at the present time, I say we don't have any difficulty.

Now, if you want to go into the question of exposure, there again I'm sorry to say to you, in spite of the doubts that some of you seem to have that we don't have any problem, because in 1941, the very first case that I processed, I personally heard it, wrote the decision. It went to the Supreme Court and I was affirmed. The evidence showed, and the medical testimony was God-awful rotten, all we had was the radiologists' reading of the film, and

his diagnosis of silicosis, and inactive tuberculosis.

Now, the State Insurance Fund didn't do anything about it so the record stood. The employer was unpredicted, and the superintendent of the mine testified that there was some silica rock in the mine and the evidence showed that that was the only place the man worked, so I said, on that record, gentlemen, this man has made a prima facie case. He has the disease, he's totally disabled, this is the only place he worked, the evidence shows that there was free silica dioxide dust exposure, so we made the award. The Supreme Court said, that's right, and our court has gone farther than that.

All of our cases, so far, come from the mine. Our Supreme Court takes judicial notice of the fact that every non-ferrous mine and every coal mine in the State of Utah has or exposes the employee to free silica dioxide dust, so we don't have any problem.

Now, we have that thirty day thing. The last employer is liable, in other words, the last employer in whose employ the employee was exposed to free silicon dioxide dust, he never takes the trouble any more to make dust counts. We don't have to go into the evidence; we don't have any problem.

BY MR. WATERS:

Jim, I'm going to ask you to take three minutes

to comment on that subject, if you will, please. For the benefit of the audience, I'll repeat the question: From your experience, what is the most difficult problem in determining contested pneumoconioses cases?

BY COMMISSIONER HILL:

I wish that I could agree with my brother, Mr. Wisely. Our biggest problem is simply this: Is the man disabled? Now, in - I can illustrate it best by a couple of typical situations.

Here is a man in his fifth decade, who has been working in a foundry for twenty or thirty years, and all of a sudden he says, I can't take it any more; I'm tired out. I'm all in, I'm short of breath walking upstairs. My chest feels kind of tight and I'm coughing all the time; so he just quits. He doesn't, he hasn't been to a lawyer. He's going to get there pretty soon.

So he starts a claim. He just says he can't work and then, of course, we're in this disputed medical again, and is that man disabled? He says he can't work. There may be some disagreement as far as the X-ray diagnosis is concerned, but frequently, they'll say he has either early silicosis, that there is some fine nodulation, but that does not interfere with his ability to work. Nevertheless, the man says he can't work and, of course, the physician who testifies for him says it would not be advisable for him to

return to work.

Then we have the situation where a man has been working a long time, probably in the same age group. He gets a stubborn cold, maybe it's bronchitis, maybe he has pleurisy. He's treated during the winter by his family physician and in the course of the treatment, the physician sends him to a radiologist and he says that he - reports that he has early silicosis, maybe some mineral fibrosis, maybe some more, so the family physician tells the man, you got dust in your lungs, Joe, you shouldn't go back to work at the same job. I'll prepare a note and you take that in to your employer when you go back to work, so he does, and the employer says, O. K., and you have the plant physician examine him, take X-rays and they say, yes, you've got a mineral silicosis, but it won't interfere with your returning to the same job. You either work at that job or no job at all.

Now, how in the world do you decide those cases? Those are just simply almost insurmountable problems, and it seems to me that to be realistic in the first illustration, here is a man with a good work record of long years and all of a sudden he quits. Are we going to say that he is not disabled, because you look at the X-ray evidence and you look at the X-ray and you say, no, this doesn't fit the pattern of the man who is supposed to be disabled from

silicosis? And in the second instance, are we going to say that the man doesn't have a right to rely on the advice of his family physician?

BY MR. WATERS:

It's a good thing that that question was not directed to a lawyer as to what he considers to be the most difficult problem dealing with contested pneumoconioses cases, because it might not reflect too much credit upon some of the Commissioners.

Gentlemen, it is indeed with regret that I must bring this panel discussion to a close, because many of the questions that have been presented, and I'm sure that many of them still in your minds, are highly controversial and I'm certain that there exist differences of opinion among us with respect to them. I think it is only fair to say that we are indebted to the Saranac Laboratory for this opportunity to receive such authoritative information and advice about these problems with which all of us must deal in the transaction of our respective businesses.

On your behalf, I wish to thank the participants in the panel for the excellent presentations that they have made and for their honest expressions of opinion and on their behalf, I wish to thank you for your interest, consideration and participation in the proceedings of the panel. I know that Doctor Vorwald and his associates in the laboratory

Chapter Thirty-four

Summary of the Symposium

Arthur J. Vorwald, M. D.

During the symposium we have had the opportunity to explore and discuss common problems with the objective that industrial health be improved. A review of the papers and discussions shows numerous entries on the positive side of our hypothetical ledger but there are quite a number on the negative side also: there are many, many things which we just do not know at the present time.

We have discussed definitions and terminology and have found out that there is not uniform agreement about the concept pneumoconiosis. How broad shall the term be? Shall it be a limited one? I believe that the weight of opinion revealed by the discussions would indicate that the concept of the dust disease pneumoconiosis should be a broad one and should not be restricted to those conditions in which significant damage to the lung has been produced by dust. Concerning the term silicosis there is some question as to whether the definition in use today is a correct one.

In reviewing old problems it was brought out that the mechanism by which free silica exerts its effect upon tissue is not definitely known. Of various theories presented, one dealing with the denaturization of protein by silica is now under investigation by the Department of Biochemistry of the Trudeau Foundation. Mention was made of divergent opinions concerning asbestosis, which indicated that observations about individuals in the asbestos industry in Canada and in the

United States ^{do} ~~did~~ not agree with similar observations about workers in the industry in England. Dr. Hardy showed that beryllium still presents many problems and that even today we are not sure that beryllium is the only factor in the disease associated with exposure to that element. Dr. Hardy believes that there is perhaps a precipitating factor; it will be recalled that many years ago Dr. Gardner suggested that there might be a factor "x". Regarding Shaver's disease there was, from the evidence presented, at least an implication that the aluminum component of the bauxite produces the disease. Production of the new synthetic silicas, the ultramicroscopic silica particles, was shown to impose many difficulties for the physician, for the industrial hygienist who has to detect these substances in the atmosphere, and for our colleagues in the legal profession who must administer laws which refer to disease caused by particles of microscopic rather than ultramicroscopic size.

An entire day was devoted to subjects related to pneumoconiosis in coal miners, during which we had the benefit of the opinions and observations of our colleagues from Great Britain. It was brought out that we do not know whether the carbonaceous component of coal, per se, is damaging to pulmonary tissue or whether the coal dust and the accompanying damage seen in the lungs of coal workers necessitate the presence of some substance other than the carbonaceous component of coal. There were discussions regarding radiological classification of coal miners and standardization in the interpretation of shadows present in the roentgenogram, and it was made obvious to all of us that the role of the radiologist is indeed a difficult one. The talks reveal^d also that the epidemiology of pneumoconiosis in coal workers appears to be more advanced in England than in the United States.

The discussions on pulmonary cancer disclosed that there are relatively few industrial substances which definitely have the capacity to produce cancer of the lung but, as Dr. Hueper pointed out, there are many materials which certainly should be watched through the years and which are indeed potentially hazardous. We were informed that the clinical evidence classifying chromate as a carcinogen is not too well-defined and that the evidence from Dr. Baetjer's experimental studies, to date, is negative. We were told about the experimental production of pulmonary cancer by beryllium but it was noted that the association of cancer and beryllium was an observation that applied only to experimental animals and should not be transferred to human subjects, pending validation of the observations on experimental animals and pending proof of an increased incidence of pulmonary cancer in individuals who have been exposed to beryllium.

There was an extensive discussion of pulmonary disability, partial and total disability, pulmonary function and dysfunction, breathlessness; all those things were mentioned. It is evident that from the physiologist's standpoint there has been much improvement in technic and methodology for establishing whether pulmonary disability exists, but irrespective of all those refined techniques the evidence presented by the physiologists today is somewhat disturbing. I feel sure that clinicians, administrators of industry and members of the legal profession are not yet willing to accept the implication that there is often a degree of pulmonary disability which we have not realized would be . I hasten to point out that dust exposures do not, in every single worker, give rise to a pulmonary disability, that industrial workers are a part of the general population and, as individual persons, are subject to those pulmonary diseases which afflict also members of the general population who have had no exposure to dust. It is

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cases of that kind that constitute a problem when determining whether an individual's disease is the result of his occupation or whether it has no relation to his occupation.

Finally, I believe that the symposium has provided the opportunity for the exchange of ideas and the discussion of problems in which we have a mutual interest and I am grateful to all who attended our meetings.

DR. ORENSTEIN: On behalf of the overseas participants in this symposium, all of whom I am sure share my views, I wish to express our appreciation of the opportunity of attending the symposium. Our problems are perhaps not the problems of the various administrations of American men in the United States. The symposium brought out one more what previous symposiums have done: that the problems of pneumoconiosis are extremely complicated; that whether one deals with the etiology of the disease, with the various ways in which its effects could be ameliorated or prevented, with compensation problems or with the administration of workmen's compensation laws, one has a feeling of pushing against an elastic wall which, as one thrusts it aside, comes back with almost equal force. But gradually we are making progress, and a symposium such as this one by posing problems, which is the first step in the solution of any problem, helps the forward march of knowledge. For the opportunity of participating in this symposium, even in the role of listeners, we are deeply grateful to all those who have organized the symposium and have worked so hard to make it a success, and in particular to Dr. Arthur J. Vorwald and to his staff.

Comment by Dr. Orenstein

are deeply indebted to you and to the participants in the program for the job that they have done in their contributions, and now, Doctor Vorwald.

BY DOCTOR VORWALD:

The end of this meeting is not easy to come before you as tired as I am and as tired as I know all of you are. I shall attempt to review the highlights of the symposium, to express to you my pleasure and the pleasure of all of us in having you with us during the course of the week.

At the outset of the symposium, I quoted from my foreword on your agenda, that at the symposium representatives of each of varying interests would have the opportunity to explore and to discuss common problems with the objective that industrial health be improved.

During the course of the week, we have had that opportunity to explore and to discuss and if we review the course of the week, we will find that the positive side of our hypothetical ledger is full, but I am distressed, as I am sure many of you are, by the negative side of that ledger. There are many, many things which we just do not know at the present time.

We have had discussion about definitions and terminology, even there there is not uniform agreement as to the concept of pneumoconioses. How broad shall it be? Shall it be limited? I think that the weight of opinion at

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the discussions would indicate that the concept of the dust disease, the concept of pneumoconiosis should be a broad one, that it should not have reference only to those dusts which produce significant damage to the lung.

We have had discussions about the term silica, silicosis, what is silicosis. As our discussions have proceeded, is our definition of today a correct one? On that afternoon, there were general reviews with respect to some old problems, problems pertaining to silicosis and we attempted to show there that these - the mechanism, the mechanics or the method whereby free crystalline silica exerts its effect upon tissue, is not as yet definitely known. Varied theories were presented in even a more recent one, that which has emanated from our own department of Biochemistry under Doctor Sheean.

We have heard about asbestos, that there too, there are some differences of opinion, particularly between arbitrations which have been brought about or come about from patients, subjects, studies of industry in this country, as opposed to those in England.

Beryllium, as it was presented by Doctor Hardy, poses still many problems. We are not quite sure even today that beryllium is the only factor, as Doctor Hardy mentioned. She believes that there is perhaps a precipitating factor. The Factor X, which was forwarded by Doctor Gardner a good

many years ago.

With bauxite, it is - there is implication at least that the aluminum component of bauxite produces disease. What those implications are, I leave to your judgment.

The new synthetic silicas, by Doctor Pratt, the new horizons in that regard, which are complicated by many problems, the production by industry of ultra-microscopic particles which impose many, many difficulties, not only for the physician but also for the industrial hygienist who must detect those substances in the atmosphere, and also for our legal colleagues who must administer laws having reference to disease which might result from those older microscopic particles.

Then the next day we had a whole day of discussion on coal and we had the benefit of the opinions and observations and studies by our colleagues from England, but even there, we are not quite sure that was coal dust. We today do not know whether the carbonation component of coal, per se, is damaging to the pulmonary tissue or whether coal dust and the resulting or the damage seen in the lungs of coal workers, necessitates the presence of some other substance other than the carbonation component of coal.

We had discussions with respect to radiological classification, an attempt to introduce standardization in

the reading of shadows which are present in the Roentgenogram. I think it was obvious to all of us that the role of the radiologists, is indeed a difficult one. Visualize, if you will, the radiologist who must read shadows in the Roentgenogram and interpret those shadows. Can you, standing on the curbstone, watching the parade, and can you see the shadow of two men cast on the street, and look at those shadows and tell me which one is black and which one is white? Which one is twenty years of age and which one might be thirty? Which one has good pulmonary function and which one does not? I think, in a sense, the problems facing the radiologist are somewhat comparable.

The epidemiology of coal workers is more advanced in England than in our own country. We have heard about the problems pertaining to the coal miners in this country, but there I must admit that the - those, the problems pertaining to the coal miners and the coal mines in our country, have not been reviewed as avidly as they have been in England. In consequence, there are many areas in the coal mining enterprise which are not defined as or with respect to their problems.

Pulmonary cancer, you will recall the discussions concerning pulmonary cancer, that in the final analysis, there are relatively few substances in industry which are definitely, definitely have the capacity to produce cancer

of the lung, but in the words of Doctor Hueper, there are many which certainly should be watched through the course of the years, which are indeed potentially hazardous.

We heard about the chromates, that the clinical evidence with respect to chromate being a carcinogen, is not too well defined, and that certainly from the experimental evidence of Doctor Baetjer, that evidence to date is negative. We have heard about the experimental production of pulmonary cancer by beryllium, but that is only an observation in experimental animals, and certainly should not have reference to human subjects, pending validation of those observations in experimental animals and pending the discovery of an incidence in individuals who have been exposed to beryllium.

The next day we had a very good discussion on pulmonary disabilities, disability, pulmonary function, dysfunction, partial and total disability, breathlessness, loss of faculty, all of those things were mentioned; which one shall we accept?

Certainly, I think it is evident that from the physiologist's standpoint, there has been much improvement in techniques and methodology and design for establishing whether pulmonary disability exists, but irrespective of all those refined techniques, the evidence presented by the physiologists today, is somewhat disturbing. I'm sure that

after clinicians, the administrators of industry, the legal profession, are not willing as yet to accept those implications that there is often a degree of pulmonary disability which we have not realized would be free, and I hasten to add also that the evidence to date points to the fact that not all dust exposures in every single worker gives rise to a pulmonary disability, that we must remember that industrial workers are also individuals of this large population of the world and that they, as persons, are also subject to those pulmonary diseases which afflict the general population and having no exposure to dust. It is those cases which constitute a problem when, is the disease which an individual has - when is it the result of the inhalation of dust? When is it the result of his occupation, or when is it a disease which has no reference whatsoever to his occupation? Those are the problems.

So, in summary, in final conclusion, I believe that the objectives of this symposium have been stated, that we have our exploration, that we have had our discussion. For that, I am grateful to all of you, especially to those who have sat here so long, to those who have often said so little, who have been here to many previous symposiums, who are really the experts in these problems, and I call your attention to our dear friend, Doctor Paul Richards, to Manfred Bowdich, to Raymond Hussey, and Doctor French, who

just left, they have said very little, and I had hoped that they would participate more than they did in the program of our symposium. Thank you. (Applause).

BY DOCTOR ORENSTEIN:

On behalf of the overseas participants in this symposium, many of whom have already gone, but who I'm sure would share my views, I wish to express our appreciation of the opportunity of attending the symposium. Our problems are perhaps not the problems of the various administrations of American men in this United States.

The symposium brought out once more what previous symposia have done, that the problems of pneumoconioses are extremely complicated; that, at first sight, whether one deals with the etiology, the history of the disease or the various ways in which its effects could be prevented and ameliorated, the compensation and the administration of workmen's compensation laws, one feels that one is almost up against an elastic wall which, as one pushes aside, comes back, with almost equal force, but gradually, we are beginning to see daylight, and a symposium such as this, by posing the problems which, after all, is the first step in the solution of any problem, helps the forward march of knowledge. For the opportunity, therefore, of participating in this symposium, even in the role of listeners, we are all deeply grateful to all of those who have organized the

symposium and who worked to - worked so hard to make it a success, and in particular to Doctor Arthur J. Vorwald and his staff. Thank you.

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(Symposium concluded 5:10 P. M. September 26, 1952).

REPORTER'S NOTE: Proper names appearing throughout the transcript, where not specifically spelled by the speakers, are subject to change or correction.

Page 137 - Reference was made to periods of early earth history. The terms in the stenographic notes appear to be Carnivorous, Rotation and Tertiary, but because of inability to check their accuracy, the blank spaces have been left to be filled in.

Page 162 - Certain German language was used which was unintelligible. The words missing would appear to refer to "pinhead" type dust deposits in pneumoconiosis.

Medical terminology and spelling has been checked, wherever possible with Dorland's American Illustrated Medical Dictionary. Some errors, however, may appear through the reporter having incorrectly heard the particular terminology.